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Publication in the JOURNAL OF PERIODONTOLOGY *is not to be understood as endorsement by the Journal or the* AMERICAN ACADEMY OF PERIODONTOLOGY *of opinions expressed in any article, abstract or extract. The object of publishing the various views is to present them without prejudice to the reader for his own evaluation.*

THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely will make the Journal a fund of extensive information, readily available to students, teachers, researchers, and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and clinical interest which are presented at the annual meetings of that society. Abstracts of important articles on periodontia that appeared during the year of 1943 and 1944 will be continued in later numbers.

We are grateful to all who have made available the subject matter from which the abstracts and copies of abstracts have been made, including the authors of the original articles, owners of the journals mentioned in the text of the abstract section, and those who have aided in any way in compiling the material comprising this issue.

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HORACE WELLS

1815—1848

THE JOURNAL OF PERIODONTOLOGY

JULY, NINETEEN HUNDRED FORTY-FOUR

1844—Anaesthesia Was Discovered, Proclaimed, and Demonstrated Through the Use of Nitrous Oxide.

IT IS especially appropriate that this issue of the *Journal of Periodontology* be dedicated to Dr. Horace Wells, because of the part contributed by a pioneer in periodontia, Dr. John M. Riggs, to Dr. Wells' discovery that nitrous oxide gas could be used to produce anaesthesia so that painless surgical operations could be performed. It was he who extracted a tooth from the mouth of Dr. Wells under anaesthesia produced by nitrous oxide gas. The first surgical operation under anaesthesia. This experiment is well described by Dr. Riggs in the following letter to Drs. Hasbrouck and Howland which was written by request. The original letter was reproduced in the *Journal of Periodontology* 10(1):12. Jan. 1939*

Drs. Hasbrouck & Howland,
956 Broadway, New York

Gentlemen:

Yours of yesterday is received and I reply, confining myself to a few facts as they occurred, illustrating the discovery of Anaesthesia.

On the 10th of December, 1844, there appeared in the Daily Times, newspaper of this City, a notice headed as follows:

"Advertisement, Laughing Gas," an exact copy of said advertisement republished in an issue of the same paper in 1870, Feb'y 17 in an article headed, "Anaesthesia in Surgery. Who discovered it?" I herewith transmit to you. It speaks for itself.

At that exhibition, Dr. Horace Wells was present and then and there conceived the idea *from an accident,* that the gas could be used to extract teeth without pain.

Dr. Colton's object was to make money and nothing else—(see advertisement).

He knew nothing of our conference that night and determination to try the gas on Wells the next morning. Wells went to the Hall and asked Colton to let him have a bag of gas, as he wanted to take it and have a tooth pulled, and he invited the party, Colton, Cooley and two others to come up and witness the operation. I was attending to a patient, but was awaiting Wells' return. When I entered Wells' office, the said parties were there. Wells took his seat in the operating chair. I examined the tooth so as to be ready to operate without delay. Wells took the bag in his lap—held the tube to his mouth and inhaled till insensibility relaxed the muscles of his arms—his hands fell on his breast—his head dropped on the head rest and I instantly passed the forceps into the mouth, onto the tooth and extracted it.

Mr. Colton, Cooley and the two others stood by the open door, ready to run out if Wells jumped up from the chair and made any hostile demonstrations. You may ask, "Why did he not get up?" Simply because he could not. Our agreement the night previous was to push the administration to a point hitherto unknown.

We knew not whether death or success confronted us. It was a *terra incognita* we were bound to explore—the result is known to the world. No one but Wells and myself *knew* to what point the inhalation was to be carried—the result was painfully problematical to us but the great law of Nature, hitherto unknown, was kind to us and a grand discovery was born into the world.

Now, Gentlemen, do not understand me as claiming *joint-discovery* with Dr. Wells. The great idea or inspiration was his—that we elaborated it together is honor enough for me.

I have no feeling in this matter except in the interest of truth, right and justice. For the credit of Dr. Colton I can say, he made the gas, gave a bag of it to Dr. Wells to experiment with—witnessed the operation of extracting the first tooth and went on his way to other cities with his "grand exhibition" and "eight strong men to make them laugh more than they have for the six months previous."

Very truly yours,

(Signed) JOHN M. RIGGS

*"Historical Background of Periodontology—Biographical Sketches" *by* Arthur H. Merritt.

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The Composition and Formation of Salivary Calculus*

By BENJAMIN TENENBAUM, B.S., D.D.S. and MAXWELL KARSHAN, PH. D.
New York City **

IT IS generally agreed by the dental profession that calculus plays an important rôle in the etiology of periodontal disease. Studies of the nature and origin of these deposits, and the conditions which determine their deposition, have not been as abundant as would be expected from their apparent importance. This subject has been reviewed in detail by Rosebury and Karshan. (22)

There are two clinical varieties of calculus, namely supragingival and subgingival. The latter is sometimes called serumal calculus. As both types of calculus are very much alike in structure (2, 3, 19) and in general composition (16, 21, 22), it has been assumed that they have a common origin, namely, the saliva. However, Box (4) has presented evidence which suggests that subgingival calculus is derived in part from exudative products that accumulate in the gingival crevice. The presence of mucin in subgingival calculus (8) would tend to support the viewpoint that saliva also plays a part in the formation of this type of deposit. Salivary calculi refer to the stones that are formed in the ducts of the salivary glands or in the glands themselves.

Supragingival calculus is deposited on the surfaces of the teeth or on prosthetic appliances which are exposed to the fluids of the mouth. It appears principally on the surfaces of the teeth opposite the exits of the ducts of the salivary glands. It is frequently present on malposed or irregular teeth, in areas difficult to reach by the toothbrush, and on the side spared from chewing because of pain or missing opposing teeth. These deposits are cream white and soft when first formed, but later harden and become stained by food and tobacco smoke. Subgingival calculus is deposited on enamel or cementum under the free margin of the gum or in a periodontal pocket. It is harder than the supragingival variety and is generally stained dark brown or black as the hemoglobin from small hemorrhages in the gingiva undergoes decomposition.

The accumulations of both types of calculus act as mechanical irritants to the gingiva and supporting structures of the teeth, and their removal by dental treatment is followed by improvement in mouth health. This would indicate that the deposits may aggravate or cause damage to the supporting tissues of the teeth making them susceptible to periodontoclasia.

Several investigators (8, 13, 16, 21, 24) have reported that supragingival and subgingival calculus, and duct and gland calculi, are similar in chemical composition, containing approximately 70 to 80 per cent of mineral matter. This consists chiefly of salts of calcium and phosphorus. The rest of the inorganic matter is mainly calcium carbonate. Salts of magnesium and other elements are present in small amounts. It may be of interest to note that the proportion of the elements calcium, magnesium, and phosphorus in calculus is quite similar to their proportion in dentin.

The deposits also contain from 15 to 25 per cent of organic matter and a little water. Included in the organic matter are the proteins mucin, keratin, and nucleoprotein (or nucleic acid) (8). Mucin comes from saliva, while keratin is derived from desquamated epithelial cells. A small amount of fatty matter is also present. Mucin which has adhesive properties helps to bind the deposit together. By decalcification of both calculus and salivary stones, Naeslund (19) was able to demonstrate the presence of an organic matrix or stroma in the deposits which was found to be interwoven with thread-like bacteria of the genera *Leptotrichia* and *Actinomyces*. Other workers (2, 3, 25) have reported similar observations.

Most of the calcium and phosphorus in

*Prepared for presentation before the Section on Periodontia of the American Dental Association, 1942.

**Department of Biological Chemistry, College of Physicians and Surgeons, and School of Dental and Oral Surgery, Columbia University.

calculus exists as calcium phosphate. To determine the type of calcium phosphate present, chemical and X-ray diffraction pattern studies have been made. It has been assumed in the chemical analysis of these bodies, that all the carbon dioxide obtained by treatment with acid originates from calcium carbonate, and that the small amount of magnesium exists as trimagnesium phosphate. After subtracting from the total calcium and phosphorus amounts of these elements to account for these salts, Karshan and Schroff (13) found in analyses of five ampules of submaxillary duct and gland calculi, an average ratio of 2.06 for residual calcium to residual phosphorus. More recently Glock and Murray (8) reported an average ratio of 1·83 for residual calcium to residual phosphorus in analyses of twenty-three samples of subgingival calculus. In tricalcium phosphate the ratio of calcium to phosphorus is 1.94. Therefore, on the basis of the ratio found for calculus, it was concluded that the inorganic matter consists chiefly of tri-calcium phosphate. However, Phillipp (20) and Mathis (18) found by X-ray diffraction pattern studies, that the bulk of the inorganic matter in calculus does not consist of tricalcium phosphate. These studies indicate that the greater part of the mineral matter is present as a complex salt similar to the naturally occurring apatite salt known as hydroxyapatite, the formula for which is $Ca_{10}(Po_4)_6(OH)_2$. More recently Glock and Murray (8), by using similar methods, came to the conclusion that calculus is in part apatite-like in structure.

In the analyses of submaxillary calculi and of subgingival calculus, the ratios of residual calcium to residual phosphorus were, with but one exception, less than that in hydroxyapatite where the Ca:P ratio is 2.15. These deviations suggest that these deposits contain in addition to hydroxyapatite one or more calcium phosphate salts having Ca:P/ratios less than 2.15.

The process by which calculus forms has been a subject of study for many years, but the problem is far from being solved. One fact is certain, and this is that saliva plays a fundamental part in the formation of supragingival deposit. To what extent this is true of subgingival calculus is still uncertain. Saliva contains, amongst other substances, a high percentage of mucin and various soluble compounds of calcium, phosphorus, and bicarbonate. In the formation of calculus, the bicarbonate and the salts of calcium, magnesium, and phosphorus, originally in solution in saliva, undergo changes by which they separate out in solid form. As these changes occur, other substances that are present in saliva, such as bacteria and food debris, become included in the solid mass. It is clear that this precipitate is not merely a sediment of saliva, since the relative amounts of the individual ingredients of saliva and calculus are very different. What happens is that a chemical change takes place which converts the soluble salts into insoluble ones; but just what is responsible for this chemical change is not yet entirely clear.

Since the main inorganic unit in calculus is a calcium phosphate salt, it is reasonable to suppose that the concentration of calcium and phosphate in saliva is related to the tendency to form these deposits. Furthermore, precipitation of calcium phosphate would also depend on the type of phosphate ion present, which in turn is dependent on the hydrogen-ion concentration of the medium. Hence a relationship may exist between pH and calculus formation. That the protein content of saliva may be related to the formation of these deposits is suggested by the work of Karlstrom and Miller (10) who found that precipitation of calcium salts was markedly reduced by the addition of soluble protein. In view of these considerations, we (26, 27) performed salivary analyses on a group of persons who tend to form heavy deposits of supragingival calculus, and on a group that showed little or no tendency to form such deposits, to ascertain if any differences exist in the salivas of these groups.

The calculus group included individuals who had heavy deposits on the lingual surfaces of the lower anterior teeth. Most subjects however had heavy deposits both on the lingual surfaces of the lower anterior teeth and on the buccal surfaces of the upper molars, or throughout the mouth. The calculus-free group consisted of individuals who were free from supragingival calculus, although they had not had dental prophylactic treatment for at least one year. The age range of the subjects studied was between 20 and 60 years.

Stimulated saliva was collected while the subject was chewing paraffin; unstimulated

saliva was obtained with minimum movement of the tongue, cheeks, lips, and jaw, Collections were made in three successive portions, from one to two hours after breakfast. The first portion, collected under paraffin oil, was used to determine the initial pH. The second portion was used for the analysis of total calcium, inorganic phosphate and protein. The third portion, again collected under oil, was used for a final pH determination.

The statistical method employed for the interpretation of the results was the standard procedure of dividing the difference between the means of the two groups by the standard error of the difference to determine the probability that the observed difference was not due to chance.

Mean values for stimulated and unstimulated saliva with statistical analysis of the data are given in Tables 1 and 2.

TABLE 1

Mean Values for Saliva of Calculus-free and Calculus Groups

Type of case	No. of cases	No. of sets of analyses	Age	Total Ca	Inorganic P	Protein	pH at beginning of collection of saliva	pH at end of collection of saliva
				mg. per 100 cc.				
				Stimulated saliva				
Calculus-free	34	69	29.6	5.1	11.9	298	7.00	7.23
Calculus	59	116	40.0	6.7	13.9	289	7.12	7.33
Difference between mean values of calculus-free and calculus groups divided by the standard error of the difference				7.2	4.1	0.6	2.6	2.3
Odds that the difference between the calculus-free and calculus groups is not due to chance				10^{11} to 1	15770 to 1	> 1 to 1	106 to 1	46 to 1

TABLE 2

Mean Values for Saliva of Calculus-free and Calculus Groups

Type of case	No. of cases	No. of sets of analyses	Age	Total Ca	Inorganic P	Protein	pH at beginning of collection of saliva	pH at end of collection of saliva
				mg. per 100 cc.				
				Unstimulated Saliva				
Calculus-free	18	32	30.2	5.6	17.1	298	6.76	6.86
Calculus	28	63	42.0	7.1	19.8	348	6.77	6.90
Difference between mean values of calculus-free and calculus groups divided by the standard error of difference				4.6	2.0	1.4	0.2	0.8
Odds that the difference between the calculus-free and calculus groups is not due to chance				> 10^5 to 1	21 to 1	5 to 1	<1 to 1	1 to 1

It is thus seen that in both unstimulated and stimulated saliva of adults, the most striking difference between the calculus-free and calculus groups appeared in the calcium content of the saliva. The mean values for calcium were lower in the calculus-free than in the calculus group, the differences being statistically significant. The mean values for inorganic phosphate were likewise lower in the calculus-free group for both types of saliva, the difference between the two groups being statistically valid in the case of stimulated saliva. For unstimulated saliva it is also likely that

the difference between the means is not due to chance. No significant differences between the groups were found for protein in either unstimulated or stimulated saliva, or for pH in the former. However, the difference between the groups for the pH of stimulated saliva may be regarded as significant.

Karshan and Rosebury (12) found similar differences in calcium and inorganic phosphate in stimulated saliva of Alaskan Eskimos, with and without calculus. Other studies (11, 14) of saliva of Greenland and Alaskan Eskimos revealed differences for calcium and inorganic phosphate in the same direction.

More recently we (28) analyzed saliva of a group of 38 patients having advanced periodontoclasia. The age range was 20 to 61 years. Ten were calculus-free, while twenty-eight had heavy deposits of tartar. Analyses were made immediately before all their remaining teeth, at least twelve in number, were extracted. Results are given in Table 3.

TABLE 3

Mean Values for Saliva of Patients with Advanced Periodontoclasia

Occurrence of Calculus	No. of cases	Total Ca	Inorganic P	Protein	pH at beginning of collection of saliva	pH at end of collection of saliva	CO_2 . . Combining power cc./100cc.	Minutes to collect 10 cc.
		mg. per 100 cc.						
				Unstimulated Saliva				
Calculus free	10	5.6	17.5	383	6.65	6.86	6.80	34.5
Calculus	28	7.0	19.7	394	6.54	6.91	7.56	30.4
				Stimulated Saliva				
Calculus free	10	5.2	11.3	280	7.20	7.49	29.6	8.9
Calculus	28	6.7	13.5	301	7.20	7.46	29.1	9.0

These findings as a whole indicate that the average concentrations of total calcium and inorganic phosphate in both stimulated and unstimulated saliva of persons who form calculus are relatively such as to favor the precipitation of the calcium phosphate found in the deposit. This holds in the presence or absence of periodontoclasia.

Since the possibility existed that particles of calculus may have been added to saliva during collection, thus affecting its calcium and the inorganic phosphate contents, analyses of unstimulated and stimulated saliva were made in eleven subjects, before and after dental prophylaxis. The average values showed little difference before and after prophylaxis indicating that the findings reflect the tendency toward calculus formation rather than the effects of the presence of calculus.

Other hypotheses that have been advanced to explain the formation of calculus have been based on experiments in which calculus-like masses were produced *in vitro*.

The view that bacteria and fungi may play an important part in the formation of calculus has been advanced by several investigators (3, 4, 19). The studies of Naeslund (19) suggest that local increases in alkalinity of saliva brought about by bacterial action may be a factor of importance. Such an increase in alkalinity would result in precipitation of insoluble calcium phosphates. Furthermore, it was shown by Bull-

eid (5), Naeslund (19), and others (3, 6, 9), that calculus-like desposits could be formed when extracted teeth are placed in saliva incubated at 37° C. When the saliva was disinfected by the addition of either phenol, thymol, or chloroform, little or no deposit appeared. How the microörganisms operate in the formation of calculus, aside from the possible local increase in pH, is not entirely clear. Naeslund (19) suggested that these bacteria may act also by decomposing proteins in saliva which tend to keep calcium salts in solution. This suggestion has been given some weight by the findings of Karlstrom and Miller (10) that precipitation of calcium salts is markedly reduced by the addition of soluble protein. Prinz (21) also suggested that the colloids of saliva play a part in the formation of calculus, but his explanation differs from that of Naeslund. He claimed that the colloids of stagnant saliva become concentrated at its surface, and that as a result of the loss of protective colloid effect, precipitation of calcium salts occurs.

Another hypothesis that attempts to account for the formation of calculus involves the enzyme phosphatase which is capable of liberating phosphate ions from organic phosphate compounds in saliva. Adamson (1) found this enzyme in human gum tissue. He was able to form calculus on silk threads which were suspended in tubes containing saliva to which a gingival extract from a dog had been added. When the extract was boiled or in the absence of the extract, no deposit was formed. He therefore concluded that phosphatase, liberated from the gums by trauma, may give rise to calculus. Recently Zander (29) identified phosphatase in the subepithelial gingival tissues and in mouth bacteria. He was unable to find phosphatase in the desquamated epithelial cells of saliva as claimed by Smith (23). According to Zander, the phophatase in the saliva of normal individuals is derived from the oral bacteria. This may help to explain the close association between calculus and the filamentous bacteria mentioned previously.

Another view is that of Kirk (15) who claimed that loss of carbon dioxide from saliva was the only essential change necessary for the desposition of calculus. However, Clark and Carter (7) have shown that samples of saliva may stand for several hours without showing appreciable changes in CO_2 content. There is no doubt that loss of CO_2 occurs on exposure because of the great difference in CO_2 tension between saliva and the atmosphere. But this loss is made good, probably by the action of enzymes in the saliva. Furthermore, it is questionable whether deposition of calculus in the mouth is dependent upon stagnation such as occurs in test tube experiments, for calculus generally forms on tooth surfaces nearest to the orifices of the salivary glands This location rather suggests a free flow of saliva with minimum chance for carbon dioxide loss and bacterial action.

The most direct evidence thus far to account for the formation of calculus is based on salivary analysis. It is known that the characteristics of saliva vary in different individuals. This would lead one to expect that the tendency to form calculus may be related to these variations. Experiments have shown that in a group of persons who had heavy deposits of supragingival calculus, higher mean values were found for calcium and phosphorus than in a group which showed no tendency to form such deposits. The higher content of calcium and phosphorus in calculus-forming groups may be related to a greater tendency to precipitate the calcium phosphate present in calculus. This tendency would be augmented by factors which cause an increase in the concentration of phosphate ions. This could be brought about by an increase in pH or by the action of phosphatase.

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Some Cementum Problems*

By BERNARD GOTTLIEB, *Dallas, Texas*

INTRODUCTION

THE CEMENTUM is the only tissue of the tooth which is responsible for the connection of the tooth with the body, particularly with the surrounding connective tissue. A completely calcified cementum surface is less able to connect itself with the fibres of the periodontal membrane than one which is not calcified and of recent formation. The same relationship applies to the epithelium. The teeth maintain holes in the epithelium and the surrounding epithelial attachment strives to close these holes, thus tending to eliminate the teeth. After the extraction of a tooth that epithelium closes the hole in a short period. The problem is not, what makes the epithelium grow down, but what prevents it from growing down. It appears that the quality of the tooth surface opposite the epithelial attachment represents that barrier. A non-calcified cementum appears to be a strong barrier and to represent the vitality of a tooth, governing its functional ability.

MATERIAL

A considerable number of dog teeth were available from root canal experiments. Only the periapical areas had been studied previously. The areas around the epithelial attachments were studied in a number of dogs' teeth. Many teeth of the same dog were available. For this study, only teeth were used where effects from the root canal manipulations on the area of the epithelial attachment could be entirely excluded.

OBSERVATIONS

In Fig. 1 the base of the crevice [sulcus] is at the enamel (a), outside the illustration. The epithelium (b) is attached to the enamel as well as to the cementum. It has finger-like extensions into the connective tissue. The cementum layer to which the epithelium is attached is thin, black, and its continuation between the thicker cementum and the dentin can be observed (c). Below the epithelial attachment a thicker layer of cementum (d) begins which is lighter than the black strip beneath. At its surface a thin strip of uncalcified cementum (e) is deposited. The epithelial downgrowth is limited to the thin, black strip of cementum.

Figures 2 and 3 are from the same tooth. Fig. 2 shows the epithelium (a) attached partly to the enamel (b) and partly to the cementum. Here, beginning at the enamel a dark line (c) runs along the surface of the cementum to which the epithelium is attached. It is less distinct outside the epithelial attachment. Outside of that line

* From the Research Department of Baylor University Dental College, *Dallas, Texas.*

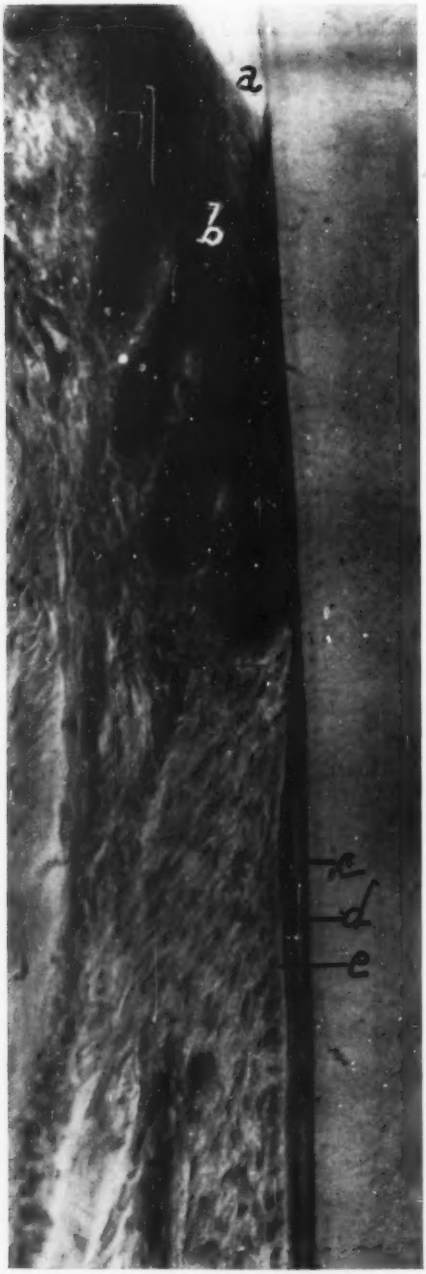

Fig. 1. Downgrowth of the epithelium (b) along a thin, dark cementum layer (c). (a) enamel. (d) lighter calcified cementum. (e) thin layer of uncalcified cementum.

only a trace of an uncalcified cementum strip is to be seen outside the epithelium. The method of downgrowth of the epithelium along the cementum is typical, the deepest point of the epithelium being attached to the tooth surface. Fig. 3 shows a part of the root surface nearer to the apex. The cementum (a) is thick, the lacunae (b) are empty having contained cells previously. The dark longitudinal lines (c) indicate an interruption of the continuous deposition of cementum, and higher calcification of that surface. Such a dark line borders the cementum to the surrounding connective tissue (d). It indicates that new deposition of cementum at the present surface was not performed. In some places a trace of uncalcified cementum may be observed.

Fig. 4 shows the epithelium (a) attached to the enamel (b). A thin dark line of cementum (c) runs along the surface of the dentin as in Fig. 1. But here in Fig. 4 we see a broad band of light, uncalcified cementum deposited on the calcified one. The width of that strip is greater over the thin strip beneath the epithelium than over the broader uncalcified cementum. The periodontal fibres (d) are ideally arranged and fixed to the uncalcified strip of light cementum. Fig. 5 shows the epithelial attachment (a) at the enamel (b). Beneath the epithelium there is quite a knob of uncalcified cementum (c). It continues into a thinner layer (d) of uncalcified cementum over a thicker layer of calicified cementum (e). This tooth was ankylosed with the bone due to heat action in the canal in connection with the root canal experiment. Comparing the arrangement of these periodontal fibres with those in Fig. 4 we observe that their functional arrangment is poor.

Quite a unique finding is shown in Fig. 6. The base of the crevice is still at the enamel, near (a). The epithelial attachment (b) is short. Contrary to the usual form (*see* Fig. 2) the epithelium is not attached with the deepest point to the cementum. The deepest point of the epithelium (b) is outside the cementum surface. This suggests that there was a stimulus to epithelial growth but something prevented it from growing along the root surface. Between the epithelium and the surface of the cementum there is here like in any other case no uncalcified cementum. At the end of the epithelial attachment (c) there begins

a broad layer of uncalcified cementum (d). That appears to be the barrier against downgrowth of the epithelium along the cementum surface. The findings described in the previous illustrations could be observed also, in the other teeth of the same dog. But such findings are not unusual. The finding described in Fig. 6 is quite unique. It could be found in nearly all teeth of this dog. That proves that some common cause produced it.

Figure 7 shows a similar condition resulting from an ankylosis caused by experimental traumatic occlusion in a dog. The base of the crevice (sulcus) is at the enamel (a). In connection with the pressure of the traumatic occlusion (a cemented crown) the root was resorbed to the line (b). The resorption undermined the enamel (c) and was repaired by the ankylosing bone (d). It appears that a stimulation of epithelial growth was present that caused the formation of the epithelial pearl (e). The epithelium seems to have been prevented from growing along the bone like we are accustomed to seeing downgrowth of the epithelium along the cementum.

Another instructive illustration is Fig. 8. An incisor of a dog was deeply broken, trying to extract it for experimental purposes. The broken root was then covered by soft tissue. The histological examination revealed that epithelium (a) isolated a great part of the fractured dentin (b) from the surrounding connective tissue (c). At one place the epithelium attached itself to the fractured dentin surface (d), leaving a part of it (e) united with the connective tissue. The epithelium did not grow all over the fractured dentin surface.

DISCUSSION

In different dogs, different conditions of the uncalcified cementum layer on the root surface were found, some with a poor cementoid layer (Figs. 2 and 3), others with a medium uncalcified layer (Fig. 1) and finally some with quite voluminous strips (Figs. 4, 5, and 6). The findings beneath the epithelial attachment correspond with the findings in the more apical parts concerning the presence of a layer of uncalcified cementum. The thickness of the calcified cementum represents history. Only the condition of the surface coat counts. In Fig. 3 we have a considerable layer of calcified cementum with empty cell holes. That

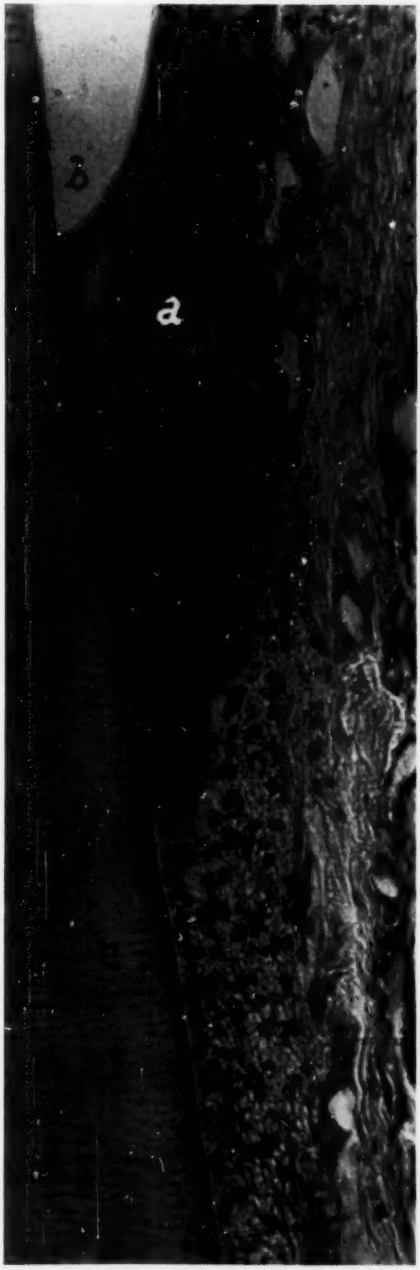

Fig. 2. Epithelium (a) attached partly to the enamel (b) partly to the cementum. The outer strip of the cementum formed by a dark line (c). No layer of uncalcified cementum above.

indicates loss of vitality of that tissue. On the surface we find a dark resting line indicating higher calcification without new deposition. The ability of the old, calcified cementum to maintain desirable connection with the surrounding connective tissue becomes doubtful. The *clinical prognosis* will accordingly be different. In cases like Figs.

Fig. 3. Same tooth and similar conditions like in Fig. 2, at an apical part. The cementum holes (b) are mostly empty. (a) cementum (c) and (d) resting lines with higher calcification.

2 and 3 a considerable downgrowth of the epithelium may develop at any time producing the denudation or pocket formation according to the topographical conditions. In cases like in Figs. 4, 5, and 6 neither unhygienic conditions nor other injuries will be able to produce a further downgrowth of the epithelium as long as these conditions of uncalcified cementum persist. The fact that the different conditions of the cementum surface described were equal in nearly all teeth of the same dog, indicates that some systemic influence is responsible for the presence and extent of an uncalcified cementum layer. That helps us understand why in extreme cases either all teeth are affected by a pathological downgrowth of the epithelium or all teeth exhibit an excessive resistance against downgrowth of the epithelium.

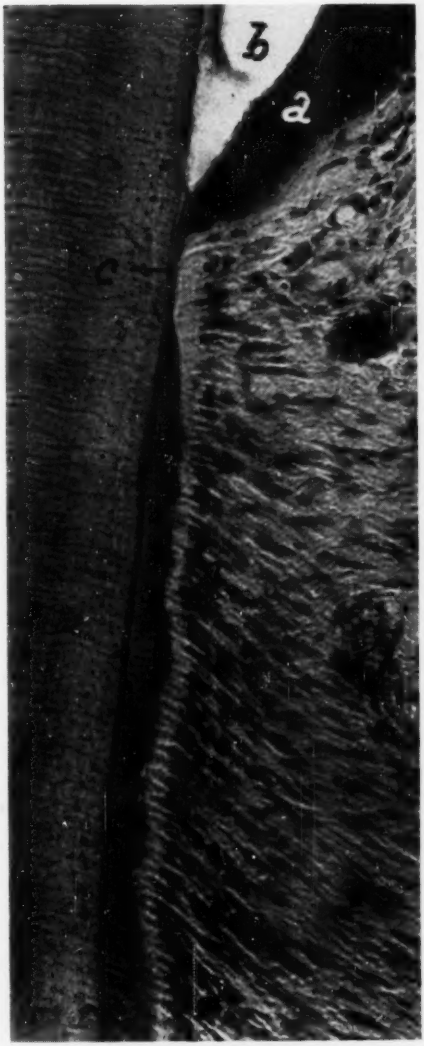

Fig. 4. Epithelium (a) attached to the enamel (b). Over the dark cementum line (c) thicker uncalcified cementum layer than over the thicker calcified cementum apically. (d) attached fibres.

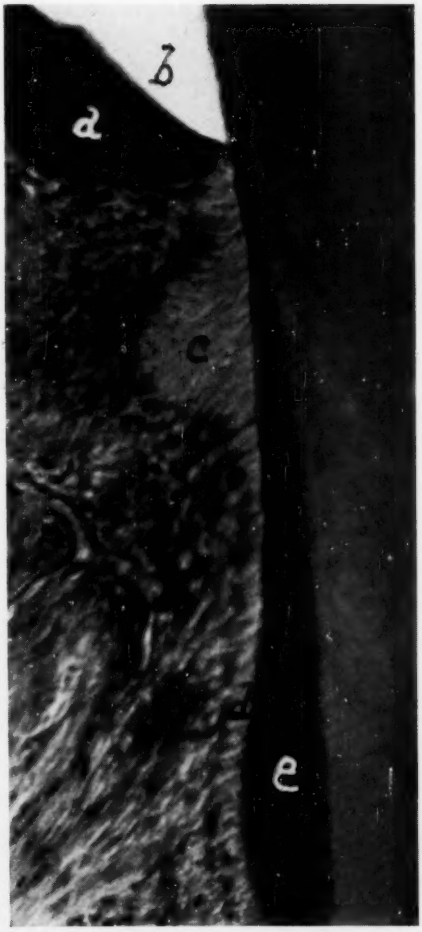

Fig. 5. An ankylosed tooth. Epi helium (a) attached to the enamel (b). Over the calcified cementum (e) a layer of uncalcified cementum (d) forming a knob (c) below the epithelial attachment.

The Figures 6 and 7 give information about the relationship of epithelial growth to its elimination capacity of tooth parts; in both illustrations there are indications of the presence of a stimulus for epithelial growth. Figure 6 shows the unique finding that the epithelium did not succeed in growing along the cementum surface, like we usually see it (Fig. 2), but grew outside of it. Here it is apparent that the layer of uncalcified cementum forms a barrier that cannot be overgrown by epithelium. Among the thousands of epithelial attachments to the tooth which we have examined in the last decade we could not find a single one where an epithelial attachment did not stop at the contact with a layer of uncalcified cementum but grew over it.

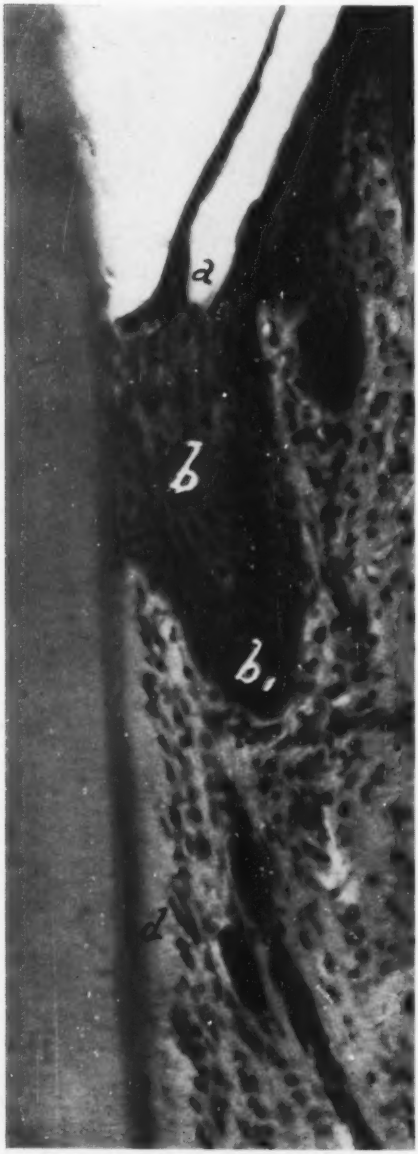

Fig. 6. (a) Base of the crevice [sulcus]. Short epithelial a tachment (b) with downgrowth outside the cementum surface (b1). Beginning at (c) broad layer or uncalcified cementum (d).

The growing of epithelium over a portion of hard tooth surface means elimination of that part from union with the connective tissue. It seems that the quality of the overgrown hard tissue plays a decisive part, though we have as yet no way to determine accurately which quality induces the epithelium to grow down. But we know a part of the story. Uncalcified cementum cannot be eliminated by epithelial downgrowth just as it cannot be eliminated by resorption. It seems that it has too high a vitality lying so near to the vital connective tissue. It appears that the elimination of a portion of the cementum surface by overgrowth of epithelium is not the cause of severing its union with the connective tissue but rather is the result of it. The disconnection with the connective tissue is primary, apparently resulting from the loss of vital qualities by the hard tissue surface which are necessary for maintaining union with the connective tissue. The elimination by available epithelium is the consequence of that loss of vitality.

It is quite a temptation to assume the epithelial attachment is prevented from progressive growth along the root surface by the barrier of fibres attached to the tooth surface. The normal specimens at our disposal for checking that possibility cannot be used to full advantage. Whenever we encounter a newly deposited layer of cementum in the presence of tooth function, we find well imbedded fibres like in Fig. 4. The new deposition of cementum means new possibilities for attaching of periodontal fibres. Fig. 7 shows prevention of downgrowth of epithelium in the absence of such fibres. But the periosteum attached to the bone may be looked upon as the barrier

proper. Fig. 8 shows a fractured dentin, not having any attachment to the surrounding connective tissue. Here we see the epithelium attached to the surface of the fractured dentin, leaving a part of it in contact with the connective tissue without eliminating it by epithelial downgrowth. Thus it seems probable that also calcified hard tissue may possess different qualities making one part ripe for elimination by epithelium and another part not. The same seems to apply to resorption by osteoclasts.

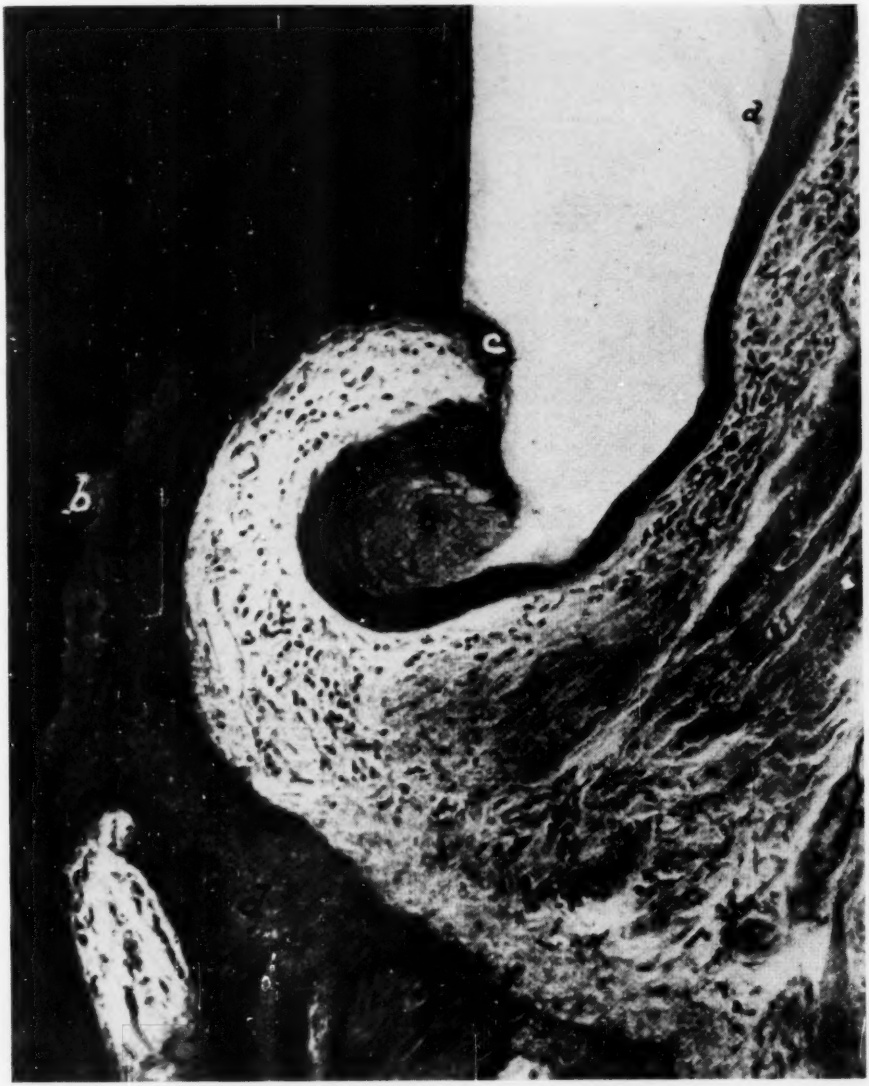

Fig. 7. Traumatic occlusion. Resorption into dentin (b) and enamel (c) repaired by ankylosing bone (d). (a) Base of crevice [sulcus]. Stimulation of epithelial growth produced epithelial pearl (e) but no downgrowth along the hard surface.

Fig. 8. Fractured dog tooth. Epithelium (a) attached to the fractured dentin (d). (c) Connective tissue. Part of the fractured dentin (b) eliminated another part (e).

We know that the cervical part of the root is formed before the apical part. If an uncalcified layer of cementum calcifies normally and after a certain time is replaced by a freshly deposited layer we must expect that at least the cementum layer outside the alveolar margin is equally thick in its calcified part as well as in the uncalcified one. (The factors governing the thickness of the cementum inside the alveolar margin are not to be discussed here). In Fig. 4 we find a strip of uncalcified cementum over the black line of a calcified one. Following that black line over the dentin apically we find between it and the light uncalcified strip a thick grayish layer of calcified cementum. The uncalcified layer is thinner than the uncalcified strip right below the epithelial attachment. Ie seems that in some cases the deposited cementum below the epithelial attachment remains longer in the uncalcified stage than in the deeper parts. It might be that the maintenance of a reliable barrier against downgrowth of the epithelium is intended. We cannot offer any attempt at an explanation for the mechanism which lies behind that teleological purpose. Findings of excessive deposition of uncalcified cementum, like shown in Fig. 5 underline such tendency. It has been shown (Gottlieb, *J.A.D.A.* 30:842, 1943) that the presence of a knob of uncalcified cementum below the epithelial attachment is the rule in squirrel teeth. From such findings we might deduct a plan for the prevention of pyorrhea (periodontoclasia): produce a barrier of uncalified cementum below the epithelial attachment, wherever it might be situated, thus preventing further progress of downgrowth of the epithelium.

It might be useful to point out that continuous deposition of new cementum, insuring connection with the surrounding connective tissue is entirely independent of function. Figure 5 is from a tooth which was entirely immobilized by ankylosis, caused by certain manipulations in connection with the root canal experiment. The presence of uncalcified cementum can be called ideal. The relatively poor arrangement of the periodontal fibres as compared with Fig. 4 is apparently to be attributed to the elimination of mobility, caused by the ankylosis, making the fixing fibres superfluous. The deposition of new cementum layers and the speed of their calcification appears to depend only on metabolic factors, which still have to be studied. Thus we may understand how some teeth with ideal function become loose and lost and teeth without antagonist for a great number of years remain firm. Only the presence of a young cementum coat counts.

SUMMARY

1 The uncalcified layer on the surface of the cementum forms a barrier against downgrowth of the epithelium.
2 The cementum immediately beneath the

epithelium seems sometimes to remain uncalcified longer than that more apically. Thus it forms a more reliable barrier. An explanation cannot be given.

3 The quality of the cementum surface seems to represent the barrier against elimination by the epithelium and not the attached fibres.

4 The immobilization of a tooth or other changes in function do not interfere with the continuous deposition of cementum. Function influences only the periodontal fibres.

5 The presence of a well-developed or a poor coat of uncalcified cementum on the root surface is equally present in nearly all teeth of the same individual. That suggests a connection with systemic conditions, which need further study.

6 Future prophylaxis will consist of production of a cementum barrier preventing the epithelium from progressive elimination of additional parts of the tooth.

NOTE—The photomicrographs were made by Charles Ervin Arnold, *Dallas, Texas.*

Wanted: A Textbook of Dental Physiology

By PAUL R. STILLMAN, D.D.S., F.A.A.P.

Longwood, Florida

THE American Academy of Peridontology is now old enough so that its history may be discussed. Among its founders were a group of dentists who had met each other as a study class under the instruction of a dentist who had devised a very complete set of scalers. They had previously recognized that persistent pus-flow was a symptom of a disease, then identified as "pyorrhea alveolaris." Other dentists therefore named these practitioners "pyorrhea specialists." That designation was objectionable.

In response to a need for further study they met and organized themselves into a permanent dental society. Recognizing that the problem of nomenclature was fundamental, their first action was to decide on a name for this society; the name finally adopted was that by which the organization is now known,—the American Academy of Periodontology. This name was coined to identify a science. That action also provided a name to identify the art as a companion for the science, giving as well a new term to replace the unsuitable "pyorrhea." This group then became recognized as "periodontists."

The nomenclature of this science was developed systematically by synthesis, in much the same way that a language is developed. It represented a new principle in dentistry. Certain corruptions have since developed through careless diction, but on the whole the original nomenclature has been preserved. At least the term "pyorrhea" is now virtually obsolete.

To adopt a name for a science-subject related to dentistry and to have it endure and become a part of dentistry's nomenclature was an achievement for the Academy. All scholastics who knew Latin and Greek were able to understand the meaning of the nomenclature proposed by the Academy. Its effect upon the dentist was in part cultural.

But let us go back a little. A science called "Dental Anatomy" had emerged, just a short time before the Academy was founded. It was the first dental science to come into existence and it was very difficult for dentists to comprehend its purpose.

Actually the first dental subject was dental prosthesis but that was not a science subject. The term "prosthesis" as applied in dentistry was adopted as a synonym or a replacement term for "Mechanical Dentistry" at a time when there was no strictly dental subject in existence. Dentists could recognize objects as exemplified in their experience with Mechanical Dentistry, but they could not recognize subjects or abstract conceptions such as Prosthetic Den-

tistry. Thus no differentiation was possible at that time. That ideological tangle had some influence upon the charter members of the Academy of Periodontology.

Science subjects had been recognized by dentists prior to the Academy's existence, but the traditional ideology of the dentist could not comprehend their purpose for dentistry. Very slowly a distinction between a science and an art was recognized by a few. That concept is not yet mature in dentistry. Our Academy was founded during the height of that confusion.

Dental Anatomy first appeared in the form of a textbook. It was accepted as a complement to dentistry rather than as a useful subject to be taught. Its adoption by the dental colleges caused a confusion regarding how it should be presented to their students. At least one college employed a sculptor to teach Dental Anatomy. Responsibility of the faculty was avoided by that expedient. The sculptor finally became discouraged and quit.

In Anatomy, as physicians comprehend that subject, a cadaver was utilized as material to represent the human organism. The cadaver is dissected subjectively in lieu of a living body, which cannot be dissected without producing a cadaver. When dentists were introduced to a cadaver, they accepted it literally, rather than figuratively, with their habit of objective thought. They soon realized that teeth could not be dissected like the vascular parts of the cadaver. That fact in itself bedimmed Dental Anatomy from the first.

Each biological subject related to the human organism adopts a theme. The theme of all Anatomy is *structure*. Physicians also have Physiology, the theme of which is *function*. As yet no textbook entitled Dental Physiology has been published.

Dental Pathology has disease as its theme. In the absence of Dental Physiology as a concept Dental Pathology is incomprehensible, as the definition of "disease" signifies. That definition reads as follows: "disturbed or abnormal physiological action in a human organism." The term "physiological action" obviously refers to *function*," rather than to the objective symptoms of disease, the method by which Dental Pathology presents disease. It thus appears that there is now a dental subject hiatus, namely, Dental Physiology.

Dental pathologists have mistaken the objective symptoms of oral disease for actual disease as the dysfunction of disease reveals. A symptom serves to point out the existence of "something else." That "something else" is the actual disease. Thus have dentists been confused by a false premise regarding the etiology of dental caries.

It would be a great honor to the Academy if Dental Physiology should emerge from within its membership. Sometime in the future Dental Physiology will exist as a definite science. That is inevitable. Let us extend the hand of fellowship to the unknown scientist who will bring it forth and greater honor if it can be imagined.

The terms "traumatic occlusion" and "occlusal trauma" are both essential to dental science and to the dental art interests of the Academy. The former has reference to an occlusion which is deficient from the perspective of Dental Physiology. The latter has reference to an injury *caused* by deficient occlusion. Here Dental Pathology provides terms and concepts through which that idea may be discussed. These terms are not interchangeable and are confusing should they merge. They are also too similar when spoken. It is suggested that a new term should be coined to replace "occlusal trauma."

To evolve Dental Physiology from the knowledges which now exist, it will first be necessary to recognize its theme. That is now known. The theme of Dental Anatomy is *structure*, that of Dental Physiology is *function*. It is the structure of the dental organ which functions. A cadaver has no dental organ in a strict sense for the reason that a cadaver has no life. A human organism has life, hence has a dental organ. This dental organ has now been recognized and its function is known; the name of its function, i.e., mastication, identifies an idea familiar to all dentists.

It would be presumptuous for anyone, regardless of his personal ambitions for renown, to hope that he could create a science. He could assemble the ideological material now in existence and compile a textbook to be named Dental Physiology. That would be honor enough.

The creation of knowledge is a very rare event in human existence. The conception that the Earth is a sphere and not a plane permitted the science of Astronomy to emerge. That was an unacceptable idea when first promulgated. Notwithstanding

all opposition, the truth found converts who built the great science of Astronomy. Truth never dies so long as human intellect endures.

It is suggested that the members of the Academy, since they are inevitably interested in the science of Dental Physiology, organize as a study group, in much the same way that the founders of the Academy did when they founded it. To state as a formula "Teeth function" is sufficient to arouse interest, for that is a truth. It is no less true than the idea "The Earth is a sphere." A grain of truth can grow to unimaginable proportions when persistently cultivated.

Several minds who are sincerely interested in any known subject can transform an immature idea by cultivating it, working together as a group. To assemble existing knowledge is far easier than attempting to create new knowledge. When all the knowledge now in existence which pertains to the subject of dental organ function is assembled, the science of Dental Physiology will have birth. Its publication as a textbook will then transform dental science and the American Academy of Periodontology if it becomes the agency of that achievement, will receive undreamed-of acclaim.

Look what is best, the best I wish in thee
This wish I have: then ten times happy me!

Notes On the 1944 A. A. P. Postgraduate Lecture Course

By Celia Rich, D.D.S., *Nashville, Tennessee*

The 1944 Postgraduate Lecture Course of the American Academy of Periodontology maintained the high standard of the three preceding years. The program followed the established plan to have these lectures feature subjects of basic importance to the comprehension of conditions which we encounter in practice, a sort of look behind the scenes of our clinical experience.

The first lecturer was the distinguished pathologist, Valy Menkin, B.S., M.A., M.D., formerly Prof. of Pathology at Harvard University, whose subject was the "Dynamics of Inflammation."

Inflammation is the complex response on the part of normal tissue to cellular damage. It may be regarded as the physical basis of infective processes. Normally the force within the capillaries balances that from the periphery, an equilibrium being established between the blood and the tissue fluids. The reaction to injury is initiated by a disturbance in the pressure *gradient,* or local fluid balance. This change is brought about or accompanied by an increase in the permeability of the capillary walls and the rapid emigration of constituents of the blood including polymorphonuclear leukocytes into the injured tissue.

Menkin investigated inflammatory exudates and found a nitrogenous substance which he calls "leukotaxine" which offers a reasonable explanation of these two basic phenomena in the inflammatory process. Leukotaxine appears to contain factors concerned with b o t h permeability and chemotaxis.

Following the first reaction the polymorphonuclears are gradually displaced by macrophages. This change seems to depend on the pH of the exudate which in turn is often referable to disturbance in local carbohydrate metabolism. When the pH falls, macrophages take the place of polymorphonuclears and they survive until the pH becomes lower than 6.8, beyond which the acidity proves fatal to all types of leucocytes, and frank suppuration ensues.

The localization of inflammation is brought about by means of lymphatic block. The "walling off" seems due to an enhanced passage of fibrinogen through the more permeable capillary wall and the formation of a fibrinous network and of thrombi occluding the draining lymphatics.

This formation of lymphatic blockade in acute inflammation plays an important role in immunity, preventing the spread of in-

fection for an interval which allows the relatively sluggish leucocytes to assemble for the purpose of phagocytosis. Bacteria disseminate through the lymphatics and the rapidity and intensity with which the lymphatic block forms when bacteria invade and injure tissue is a gauge of their invasiveness. The ability of pathogenic organisms to disseminate from their site of inoculation is apparently inversely related to the intensity of the induced injury. Staphylococci produce a violent local reaction with correspondingly rapid lymphatic blockade. *Str. hemolyticus* on the other hand produce a mild local irritation with no lymphatic fixation and are correspondingly dangerous because there is no check to their invasiveness.

While the prompt "walling off" of an infective area retards the full effect of its virulence on the host, the virulence *per se* of the pathogen remains essentially unaffected.

In addition to leukotaxine formation there is often observed an increase in leukocytes in the blood during infectious processes probably referable to the liberation in the inflamed area of a leukocytosis-promoting factor not as yet completely identified.

It is obvious that the "inflammatory reaction thus displays an extraordinarily complex mechanism tending to localize and dispose of a chemical or bacterial irritant. This ultimately leads to organization and repair of the affected tissue."

Menkin included in his second lecture a brief scholarly discussion of diabetes associated with infection, and the rationale of increased insulin administration when this complication exists.

Dr. Balint Orban's lecture in contrast to Menkin's dealt with inflammation of a specific area. Inflammation runs a regular course but the reaction is modified by the tissue involved; and the gingival sulcus is unique. Many types of irritants are always to be found here, all the conditions conducive to inflammation. And, as a matter of fact some degree of inflammation is invariably present in the gingivae. Here we may observe clinically over a period of years all the phenomena analyzed by Menkin. The gingivitis may not be severe enough to create a leukocytosis, but even the milder irritants or injuries initiate leukotaxine formation and increased capillary premeability, with migration of cells from the small vessels into the tissues. With accentuation of the irritation we may find leukocytes accumulating and later these changing to macrophages as the pH falls.

Dr. Orban presented a remarkable picture showing leukocytes actually squirming through the vessel walls. Then extremely interesting slides showed the abundance of leukocytes in Vincent's infection while in a fatal case of agranulocytosis there was acute inflammation with edema and necrosis but no leukocytes. He warned that when ulcerative gingivitis fails to respond promptly to treatment agranulocytosis may be the diagnosis and this condition is always serious and often fatal. Other photomicrograph slides showed various types and degrees of inflammation. Sometimes the surface of the gingival epithelium is inflamed while the pocket lining is healthy as may occur in skin diseases.

In chronic gingivitis plasma cells are present. Their function is not understood but they may be oxygen carriers. With the clinical application of oxygen either as Dunlop's vapor or as superoxol these plasma cells break up and disappear, oxidation of carbohydrates is increased and tissue acidity diminished. Thus, superoxol is an irritant but a specifically beneficial irritant and after its application new fibroblasts are found among the plasma cells and a decided change for the better in the inflammatory picture.

The Program Committee is to be congratulated on the arrangement of the lectures, one leading up to another which it overlaps in some respects.

While Órban's lecture dealt in the main with the histological picture of different types, degrees, and stages of periodontal disease, Dr. Hine (Maynard K. Hine, D.D.S.) presented the subject from the clinical and etiologic aspects of these disturbances.

He divides all periodontal pathology into acute and chronic which in turn he subdivides according to tissue involved and the predominant tissue reaction. We have thus gingivitis, periodontitis, and periodontosis with all their modifications. Tissues react to injury in only a limited number of ways, such as inflammation, hypertrophy, atrophy, degeneration, necrosis, etc.; but the causes of these reactions are many, the kind and degree of irritation, the time it is in effect,

and the ability of the tissue to withstand injury (tissue resistance).

This last is again dependent on local or systemic factors or both.

There are also idiopathic cases, whose etiology is undiscoverable.

Because of this variety and complexity of etiological factors, careful analysis of each case should be made if success in treatment is to be expected.

Melvin H. Knisely, M.D., of the Department of Anatomy of the University of Chicago showed remarkable motion pictures of microscopic circulatory pathology, further clarifying the mechanism of these phenomena.

The method of making these pictures is itself most interesting and it has simplified experimental research. Dr. Knisely first identified for us normal capillary circulation. Each tissue has its own rate of blood flow essential for its supply of oxygen and other food. A lack of oxygen injures cells and in response to the injury leukotaxine is formed and the "leaking" of blood vessels is increased.

The film showed the microscopic circulation of living rhesus monkeys during acute inoculated Plasmodium knowlsi malaria. The work was done through the coöperation of the University of Chicago and the University of Tennessee Medical school.

The first film in the sequence introduces the method of studying the circulation in living animals contrasting normal and pathological conditions. The second traces the course of the disease in the untreated animals to several types of fatal outcome. The third shows the alteration of the pathological blood through medication whereby the circulation returns "towards normal."

These pictures are most amazing. We actually saw diseased blood in circulation, the condition getting worse and then improving as we watched. This work is an introduction to the study of the many human diseases characterized by agglutinated and precipitated blood.

Maurice Seevers, Ph. D., M.D. and H. M. Pollard, M.S., M.D., Prof. of Pharmacology and Prof. of Internal Medicine, respectively, at the University of Michigan, presented a timely series of lectures on the "Pharmacology, Toxicity and Clinical Application of the Sulpha Drugs and Penicillin."

The introduction of these drugs marks a new era in the treatment of infectious diseases. Their action seems to be an interference with the metabolic processes of bacteria. In brief, they enter the bacterial cell replacing or rather substituting for para-amino-benzoic acid whose molecular structure is very similar to their own. The bacteria being unable to subsist without this acid, are thus partially starved, after which phagocytes dispose of them. In this connection it is interesting to note that certain proteins are bacterio-static and Dr. Menkin has pointed out that proteins osmose from capillaries at the site of injury, and thus we see again nature defending herself.

A great advantage is the fact that large doses can be administered against bacteria without harming the host. In fact large dosage is safest. This obviates the danger of organisms developing a resistance or "sulpha-fastness." It is better to give large amounts and taper off maintaining a pretty high level in the blood. If small amounts are given over a long period as may happen in self-medication and by chewing sulpha-gum, etc., such sensitivity may develop that when it becomes necessary to administer large amounts serious and even fatal reactions are possible. At the slightest indication of sensitization the type of sulpha drug should be changed; also if one type fails to act in a given case others should be tried.

A fortunate rule with penicillin as well as with the sulfonamides is that the more virulent the organism the more susceptible it is to these agents. It is important to know when to stop administration, but it is usually desirable to continue for some time after clinical symptoms have disappeared. This is especially important in cases of systemic *Staphylococcus* infections. There is a varying degree of activity of different drugs of this group against various types of infection but in a general way it may be said that practically all the cocci are susceptible to them. The *Str. hemolyticus, gonococci, meningococci, pneumococci, Welch's* (gas) *bacilli,* and *Staph. aureus,* are especially susceptible; but less susceptible are the anaerobic *Streptococci, Str. viridans,* and the microörganisms in influenza, malaria, and undulant fever. Apparently these drugs are quite ineffectual against typhoid and any virus disease, although these are sometimes helped by the effect of the drugs on secondary invaders.

Among the dangers accompanying sulfonamides, cyanosis must be watched for;

it is usually an early symptom and the drug should be stopped forthwith before too many erythrocytes are destroyed. The newer compounds are safer in this respect but they are still capable of destroying the white-forming centers in the bone marrow. The white count therefore should be made every few days to avoid agranulocytosis or leukopenia with possibly fatal termination. The local use of the drugs singly or in combination is considered advisable although in large wounds they may be incapsulated and become an irritant.

Kidney damage is possible from sulpha crystals but this is now avoidable by the administration of sodium bicarbonate along with the drug.

Mortality from lobar pneumonia has been reduced from 40 or 50% to 10% and many of the complications following the disease have been reduced. The sulfonamides offer much in sub-acute bacterial endocarditis.

Penicillin, Dr. Seevers considers the greatest single thing in chemotherapy. Its potency against infections combined with its low toxicity to mammalian cells are its great advantages. Certain bacteria are particularly susceptible, *gonococcus* for example, even where they are sulpha-fast. The effectiveness against *Spirochaetes* is most important especially because it obviates the high mortality rate following administration of some other powerful spirochaeticides. It is effective against *Str. hemolyticus*, but poorly so against *Str. viridans*. The dosage varies. *In vitro*, very large dilutions are effective. On the other hand systemic *Staphylococcus* infections require large dosage; *but it can cure them.*

Penicillin is ineffective against malaria but it should be remembered that penicillin itself was discovered accidentally, incident to other studies, and it is hoped that the extensive investigations now going on may develop or discover anti-malarial substances.

Malaria is the world's greatest health problem, 4/10 of the total world population having it in some degree, many in a subclinical stage, and thus far drugs have been only suppressive.

The slowness of manufacturing penicillin and the difficulty of getting a pure product are its great disadvantages.

The government is sponsoring researchers and manufacturers in attempts to synthesize penicillin, but there are many difficulties in the way not the least of which is the fact that it is extremely labile and highly hydroscopic. Most likely a new and more stable penicillin-like substance will be found.

The "Flora unit" was explained as the amount of penicillin necessary to produce bacterial stasis in a standard size colony of *Staph. aureus.*

Needless to say the benefits of the use of penicillin in the army are incalculable, the army using 85 per cent of all that is produced.

Interesting research in Vitamin K indicates that it is produced in the intestinal tract by bacteria which elaborate it from food if the bile is normal. Under these circumstances the liver apparently is stimulated to form prothrombin. Experiments have been made with direct administration of bile to produce the above physiologic phenomena.

Dr. A. C. Curtis' lecture took up the story where the foregoing left off. He presented a very pertinent discussion on Syphilis. The first manifestation of the disease is at the sight of introduction this being frequently the lips where it may often be seen first by the dentist. Even the slightest suspicion concerning sores on the lips warrants sending the patient to a syphilologist for a dark field examination. The dentist's responsibility here is great because blood examination may not show infection for several weeks but it starts disseminating at once through the lymphatics and rapidly reaches a secondary stage where the percentage of cures is greatly reduced. Eventually, after one to two years with or without treatment, the infectiousness of the disease becomes much reduced and at the end of four years it is probably non-infectious being walled off in various localities and not in the blood stream. It can however be transmitted *in utero* through the mother. Dr. Curtis traced the development of treatment from Ehrlich's to the present possibilities of penicillin which promises to be ideal. Research in syphilis has been stimulated by the war because of availability and regimentation of clinical material. Dr. Curtis thinks that various tropical diseases may prove to be different types of syphilis although in some of them the organisms may be greatly attenuated.

We knew Dr. Curtis because of his fine presentations at the two previous Seminars on vitamins and nutritional and blood deficiencies; and particularly for his interesting hospital clinics, but lack of time precluded this last feature. Dr. Curtis is now Professor of Dermatology at the Unversity of Michigan.

At the dinner meeting the first evening Commander Kerr gave a delightful and inspiring talk on the dental set-up at the Great Lakes Naval Training Station. He left no doubt in our minds that for the men under his command the dental situation is thoughtfully, intelligently and skillfully handled.

The last evening Dr. Phillip Jay of the Dental Faculty of the University of Michigan brought the group up to date on the University of Michigan's research in caries control, taking us through the laboratory and discussing in addition some of the other theories and possible factors in caries production. But he is convinced that carbohydrate intake and the *lactobacillus acidophilus* are the all-important factors in the disease.

The final lecture was given by Dr. Carl D. Camp from the University of Michigan. His subject was "Fatigue." Dr. Camp advised that it should be handled according to type, muscular, mental, or nervous. A tired feeling is not always real fatigue. It may be mere sensation distinct from any organic process. In some cases economic necessity may make it desirable to go on beyond the fatigue point. Working powers may be increased by added stimulus, such as more pay, but the effect on production especially in accuracy is rapidly detrimental. Alcohol, benzedrin and caffeine are of some help in overcoming fatigue temporarily in emergencies. Tiredness may result from insufficient nourishment and Dr. Camp thinks the British fifteen minutes for tea around 4:00 P. M. is desirable. When one is generally tired it is important to find out *why*, and to realize that fatigue may be a symptom of disease.

CLOSING COMMENTS

Dean and Mrs. Bunting again entertained us with one of their delightful afternoon parties.

Too much cannot be said of the excellent facilities offered by the University, and of the admirable way in which the course was conducted by Chairman Yeretsky.

The twenty-six students who came to attend the course represented eleven different states in widely separated sections of the country. For many it was not the first time to attend these courses sponsored by the W. K. Kellogg Foundation at the University of Michigan, and most of us intend that it shall not be our last.

The Journal of Periodontology will be published quarterly beginning with the January, 1945 issue.

Subscription price will be advanced to $4.00 annually

Editorials

Post-War Thinking

FOR some unexplainable reason, human beings most often choose to learn the hard way. One learns to value peace by experiencing war; health through illness; life by the loss of loved ones; privilege by means of deprivation; the simple lessons of life with hard knocks. Parents chide their children for not profiting by the experiences of older people but youth will have none of it. And so we repeat generation after generation the same mistakes and are individually somewhat proud of it. It is rarely that one can be taught except by personal experience even though fingers do get burned.

Peace and plenty encourage an easy way of life which extends even to one's thinking. It is much easier to give a subject superficial thought than to seek into its deeper meaning, its whys and wherefores.

American manufacturers are now promising us the last word in items of luxury "after the war." Let us not be deceived for the millennium will not come so soon. And if we are wise we will not desire it. It is during periods of stress that we are at our best. The British during the blitz and at Dunkirk; the Americans after Pearl Harbor and during the depression; the Chinese at war for seven years; the French and Dutch under Nazi occupation! Compare these peoples, any of them, in less difficult periods in their history. This is not to argue that we must suffer in order to be saved, but to suggest that more serious thinking in times of peace, health, and prosperity, might prevent some wars, much sickness, and many depressions.

Professional men and women have been trained to think, and now, if ever, it is their responsibility to do some mighty serious thinking about many things, some quite minor. For example dentists should evolve a satisfactory definition of "dentistry." Webster defines dentistry as the "art or profession of a dentist" and a "dentist" as "one whose profession is to treat the teeth and to make and insert artificial teeth." Is this a satisfactory definition of dentistry? Dr. A. E. Rowlett of Leicester, England defines "dentistry," in a recent issue of the *British Dental Journal,* as follows:

"Dentistry is an art based upon a science. It is an integral part of the Health Service of the community, being concerned directly with the teeth *and the surrounding oral tissues,** and indirectly with the whole of the rest of the body."

Other subjects demanding more than shallow thinking are; how to improve the attitude of the public toward dentistry; how to extend the benefits of dentistry to more people; how to recruit students for dentistry; through what channels can dental health education of the public be best promoted; can dentistry be placed on a production basis; are gold inlays even a large part of dentistry, and will their mass production solve any of our immediate problems?

A sense of the relativity of dentistry to other health professions and sciences, and to economic problems of the public, must be cultivated so that a proper evaluation of it can be made.

As is well known, the *demand* for dentistry bears no relation to the *need.* One basic cause for this situation is the fact that the majority of people do not

value the natural teeth and expecting to lose them anyway are not averse to getting rid of them at an early age. Instead of attempting to sell dentistry to the politicians via the assembly line, would it not be wiser to seek out ways and means of changing the attitude of the public toward dentistry, for example, by selling them oral health, instruction in better home care of the teeth, information about the dangers resulting from mouth infection, the importance of adequate nutrition, and the advantages of prevention in dentistry accompanied by the teaching of prophylactic measures. Such an approach would result in a more respectful attitude toward dentistry from various groups the lack of which we often resent. The *demand* for dentistry would then sooner tend to approximate the *need*.

—G. R. S.

* *Italics* not in original.

Post-War Plans

FOR some years, a need has been felt for more frequent issues of the *Journal of Periodontology*, in order to provide greater continuity and to stimulate further interest in periodontology. The Supplements to the regular issues were tried as an experiment and it can be stated that the increased interest in the Journal as a result has been most gratifying. It is with much pleasure that we can formally announce that the Executive Council of the American Academy of Periodontology has decided to publish the Journal quarterly beginning with the January, 1945 issue. The subscription price of necessity will be advanced. The subscription list must at least be doubled. Members of the Academy will be urged to secure at least one new subscriber each. Many might like to give a subscription to some dentist in the armed services.

The generous distribution of periodontal literature is an effective means of supplying information to those dentists who are concerned about their inability to cope with diseases of the supporting tissues of the teeth. There are still too many who sincerely believe that nothing can be done about these common afflictions.

A continuous effort will be made to improve the quality and the usefulness of the Journal. War time handicaps have unavoidably affected its physical quality, but such changes are noticeable, also, in most current dental periodicals. New features will be added from time to time and subscribers can be assured that future issues will be of permanent value to them.

In Our Opinion

VINCENT'S INFECTION—

Initial Statement

By HAROLD J. LEONARD, D.D.S., B.A., *New York, N. Y.*

VINCENT'S infection is an ulcerative disease in which necrosis of tissue occurs from the surface inward. It is believed to be caused by a group of microörganisms acting in symbiosis on tissues which are seriously debilitated. It may occur on the mucous membrane of the mouth, throat, or elsewhere. If the organisms are inhaled as in general anesthesia from a mouth or throat infection, necrotic infection of the lungs may occur. The most common place of infection is the gingiva starting with the margin of the interdental papillae just beneath the contact point. The tissue is destroyed to form a pit or crater at this point which will divide the interdental gingiva into a labial and lingual portion which may be pulled apart. A dirty gray slough covers the ulcerated surface in the acute cases.

The organisms involved are one or more of the mouth *Spirochaetes, B. fusiformis,* anaerobic *Streptococci,* and *Vibrios.* D. T. Smith has found that it required the presence of all four to produce the fulminating necrotic infections in test animals. These organisms are all present even in normal mouths so there is evidently something more needed to produce the infection than the presence of the organisms. Undoubtedly the organisms gain in virulence as they propagate in lesions in susceptible individuals. However, it has not been found possible to introduce the infection from the most severe case into the gingiva of a perfectly healthy individual. It is only contagious where there are debilitated tissues for it to attack.

The *Spirochaetes* of Vincent's disease are anaerobic. They are the organisms deepest in the lesion and are protected from the air by the layers of the other organisms outside. The infection is negatively chemotactic to P. M. N. leucocytes so that pus does not form in the lesions while Vincent's is the predominant infection. In mixed infections in which Vincent's plays a minor role, pus may form.

From the craters in the interdental papillae, the necrotic ulceration may extend to cause destruction of the papillae with concavities where once they stood. The labial and lingual gingivae may be involved and destroyed leaving the bone bare for a distance about the teeth. In severe acute cases the local infection may lead to death of the patient. The ulcerations are extremely painful especially to touch; they bleed profusely on being touched but also spontaneously; there is a peculiar very fetid odor; the lymph glands draining the area are swollen and the patient usually has a mild fever and malaise. In infants the tissues of the cheek become infected leading to extensive necrosis.

There are all degrees of severity and rapidity in Vincent's infection from the barely perceptible infection the symptoms of which come and go, but may cause gradual destruction over the years, to the severe acute which unless properly treated, may lead to death in a few days or weeks. The rate and severity depend on the general and local debility of the tissue invaded. In its chronic

state it may be present deep in periodontal pockets and be the active cause of progressive periodontoclasia. It may be recognized in this condition by the acute soreness at the base of the pocket, the profuse bleeding on probing, and by the foetid odor of the débris in the pocket. Its presence often leads to hypertrophy of the gingiva of which it is a common cause.

General debility predisposing to Vincent's infection has been found experimentally to be due to any one of many factors. Deficiency of vitamins A or D, ascorbic acid and members of the B complex, especially niacin, have been shown to cause the disease in laboratory animals. Nutritional deficiencies are believed to be the common cause in human beings. Some patients with mild chronic Vincent's infection are able to make the symptoms disappear or reappear at will by taking niacin or not taking it. Recent dietary surveys have shown that a large proportion of the population is getting suboptimum amounts of one or more of several nutrients. It is now believed that susceptibility to Vincent's infection is one of the first outward symptoms of some of these deficiencies. Recent evidence indicates that niacin deficiency with accompanying deficiencies of other members of the B group excepting thiamine is the common nutritional defect leading to Vincent's infection in human beings rather than avitaminosis A, C, or D.

Dietary deficiencies are not the only cause of general susceptibility to Vincent's infection. Confinement as in captivity, worry, overwork, illness, excessive use of alcohol, endocrine imbalances, and probably many other physiologic strains have a similar effect, the exact chemical nature of which is unknown. D. Ziskin has produced the disease on monkeys by parenteral administration of the endocrine hormone prolan, and the disease can be regularly produced in dogs by intravenous injection of Scillaren B.

Locally the resistance of the gingiva depends on its tone and the cleanliness of the teeth. It is unlikely to occur in a well cared for mouth. Indeed, it is in most instances easily cured by cleaning the teeth and instituting vigorous home care of the teeth and gums.

In the severe acute condition it is generally wise to use oxidizing washes for the local lesions and systemic therapy before undertaking instrumentation. While cleaning out the lesions by instrumentation and starting tooth brush procedure is promptly effective in cases of Vincent's gingivitis, the treatment is unduly painful and may possibly be dangerous due to bacteremia. In the severe cases it seems better to start the cleansing with oxidizing mouth washes. Agents like half strength hydrogen peroxide (1.5%) or sodium perborate solution (2%) act as cleansing agents because the catalase in the blood and tissue fluid of the ulcers decomposes the drugs with liberation of nascent oxygen, the effervescence of which cleanses the wound and introduces oxygen to the advanced anaerobic organisms. It is not a disinfectant as all microörganisms are not immediately killed by this means but it does break up the environment which permits their growth. A solution made up of four ounces of *fresh* hydrogen peroxide and four ounces of 1 to 1000 mercury bichloride solution has been found even more effective than the half strength peroxide alone. It is used by taking one teaspoonful in the mouth and flushing vigorously throughout the mouth by the pumping action of lips and cheeks for sixty seconds after which it is expectorated. The mouth should not be rinsed for five minutes but the remnants of drug should be expectorated to avoid swallowing. In the acute cases for the first three or four days the rinsing is repeated at intervals of three hours except during sleep. Gradually the time intervals are increased until it is used only at morning and night until the eight ounces of solution are used up. The pain and acute soreness will start to subside within an hour of the first using and in two or three days the instrumental cleansing and tooth brush massage may be started with safety and without undue discomfort. The drug is not sufficient to eliminate the infection completely in all cases because the drug will not reach deeply into periodontal pockets, third molar flaps, or crypts in the tonsils where infection may reside.

In a few cases the half strength hydrogen peroxide or 2% perborate may cause superficial burning of the mucous membrane especially in children. In this case it may be diluted to half the usual strength. No harm

appears to result if a trace of the peroxide-bichloride mixture is swallowed.

Many other drugs have been used in this infection with success. Indeed, nearly every author seems to have his own special remedy. Among these remedies caustics and arsenicals have the greatest vogue. Chromic acid solution is probably most popular among the caustics. The necrotic sloughs are removed mechanically either with cotton swabs or by sprays and 10 per cent chromic acid is applied to the ulcerated surfaces. The treatment is fairly successful but results are much slower than with the peroxide mouth washes and require much more time on the part of the operator.

Neoarsphenamine is used locally and systemically. Sollman *(A Manual of Pharmacology)* states that Vincent's infections "are promptly arrested by intravenous injection of neoarsphenamine 0.3 to 0.4 gm. The pain disappears in a few hours, and the inflammation subsides within a day. A second injection is rarely needed. Dry neoarsphenamine or arsphenamine dusted on the lesions is also effective." This statement by Sollman is much too positive. Many cases are not cured by these means which do respond readily to the oxidizing mouth washes followed by instrumentation and tooth brush treatment. Many cases have been seen and reported in which Vincent's infection developed during the time the patient was under treatment with neoarsphenamine for syphilis. The local application of the drug to the ulcers is painful to the patient and requires much more time on the part of the operator than the use of the oxidizing mouth washes.

Besides the local treatment, the patient needs therapy to overcome the general debility which is the essential cause of the disease. If the case is very severe, the patient should be put to bed, given a mild cathartic and be placed on a light diet with the idea of reducing physiologic strain to a minimum. A high vitamin therapy should be administered immediately using vitamin B complex capsules each containing a day's optimum quota of all known B complex nutrients plus brewers yeast for as yet undiscovered components. At least three capsules a day should be administered to build up reserves. In the meantime a patient's regular diet record should be obtained and analyzed to discover wherein the diet has been suboptimal. Dietary suggestions to make the diet completely optimal in all respects can be made after the period of vitamin therapy so as to avoid the permanent use of vitamin pills and capsules. Other causes of lowered resistance such as overwork, worry, lack of sufficient sleep, and the excessive use of alcohol should be modified so far as possible. Vincent's infection may be regarded as a first symptom of a break in health due to some underlying cause. It should not be enough merely to relieve the patient of Vincent's infection. The underlying cause should be discovered and eliminated if possible, for if not, the patient will sooner or later have other and perhaps more serious impairments of health.

EDWARD L. BALL, D.D.S.
Cincinnati, Ohio

Those of us who have limited our practices to Periodontia see many cases of Vincent's infection. Many of these are referred to us in the acute form. At the present time hardly a day passes that we do not see or treat this prevalent, miserable infection.

Obviously, we seek a cause when the number of cases increases. Let us consider then some of the factors that tend to bring on the multiplication of Vincent's infection cases.

In these days of tenseness, workers and students alike are so pushed for time that it is quite easy to develop slovenly habits of cleanliness. Students with accelerated programs fail to follow the good rule of balanced diets. The routine of life for men and women who are working on the day shifts, the swing shifts, and the graveyard shifts is undergoing a radical change. Many eat at all hours, and when they do, it is to select food wholly inadequate for sound and solid sustenance. The sales of soda pop, alcohol, and tobacco have steadily increased. These stimulants are supposed to be supplying the energy and "pick-up" for frayed nerves. There is no time to partake of the good substantial meal that is essential for strong body building and maintenance. All these physical upsets reflect themselves in the oral cavity and especially at the gingival margin.

ACUTE VINCENT'S INFECTION

For more than twenty years I have been treating Vincent's infection. In the acute

cases I have found the symptoms practically the same, i.e., sudden onset, pain, ulceration, nervousness, loss of sleep, loss of appetite, loss of weight, offensive odor, active mouth glands, metallic taste of saliva, swollen sub-maxillary glands, abnormal temperature, and not uncommonly, sore throat.

The diagnosis and treatment is comparatively easy and quick results are often dramatic. In these emergencies and during these busy times, short effective treatments are vastly important. The Vincent's cases are usually slipped in between regular appointments as time is a big factor in all dental offices. We are primarily interested in giving the patient immediate relief, and stopping the growth of the two most prevalent germs, the fusiform bacillus and Vincent's spirillum.

Treatment:

A special steel tray is used containing two glass dishes (jiggers), one-half dozen cottonoid strips, a spray bottle, several cotton applicators, one scaler, a mirror, and pliers. This tray is used only for Vincent's treatment and all contents can be, and are boiled immediately after.

The mouth is sprayed with H_2O_2. The patient is asked to rinse with warm water. Areas are isolated with the cottonoid strips. We have used the Adams treatment for over twenty years and have found nothing better. It is as follows: Churchill's idodine is used buccally, lingually, and inter-proximally, followed immediately with 10 per cent silver nitrate. Adams advocated 35 per cent $AgNo_3$ and Harold Box told me recently this strength solution should be used. The treatment is given on three consecutive days and then every other day for several treatments. Scaling and prophylactic treatment is started as soon as possible. We have found the sooner the mouth is clean the quicker we get results. The directions for home care during this treatment are written out. The routine given is as follows:

℞
1. Four large glasses of orange juice daily
2. Mouth wash of peroxide every two hours
3. No smoking
4. No alcohol
5. Rest
6. Cathartic
7. Boil dishes

In very young patients a solution of copper sulphate has been effectively used where the application of two drugs has been difficult.

I can report several cases of acute Vincent's infection that have cleared up without any drugs whatsoever, as the patients were opposed to medication. Careful prophylaxis and warm water brought about a complete and satisfactory result.

The recurrent or chronic cases of Vincent's infection or gingivitis are treated the same way. However, if the tissue will not shrink, or if the inter-proximal papillae will not connect, I do not hesitate to perform a gingivectomy. Many stubborn cases have responded beautifully to surgery. I can heartily recommend this procedure.

I see no reason for injections. Even in the most vicious cases they have proved of little advantage. After all, the most vital treatment consists in treating locally, the gingival margin. Unless this is done, we never eliminate the "nesting place" of the two most prevalent bacteria. In my opinion, where the injections are solely used, there is a positive tendency toward chronic or recurrent gingivitis.

After we have treated our cases locally, eliminating all pockets and crevices, it is absolutely necessary to maintain the shallow sulcus. Thus daily care is strongly encouraged. Much time is spent teaching the patient the exacting technique of meticulous mouth care through the use of the tooth brush, inter-dental stimulation, vigorous mouth rinsing with a warm solution of sodium bicarbonate or sodium chloride and occasionally with a solution of hydrogen peroxide.

In conclusion, let me say the only answer for "success" in the treatment of Vincent's infection is a shallow sulcus, meticulous daily care, adequate diet, moderate use of tobacco and alcohol, if at all, and regular dental supervision.

VIRGIL LOEB, M.D., D.D.S.
St. Louis, Missouri

Vincent's infection of the oral mucosa, whether it be limited to the gingiva or extended to the buccal areas, has been the subject of controversy for over twenty-five years. Much has been written on its etiology, but by far the greatest number of con-

tributions have related to its treatment. It is questionable whether any condition which the periodontist encounters, including periodontoclasia, has been subjected to as many different types of treatment. Most of the drugs in the pharmacopoeia, including the sulfonamides and penicillin, have been employed with varying results.

Dr. Leonard in his "Initial Statement" on Vincent's infection has covered the subject in a very broad way, and in it has given most of the theories as to the etiology and some of the methods of treatment. It seems to be pretty generally agreed that the actual cause is not a single organism or necessarily a group of organisms, but perhaps a filterable virus which has the power of rendering virulent many of those organisms which are commonly found in the oral cavity. Unquestionably, lack of oral hygiene is a predisposing cause, for the disease is found more often in neglected mouths. One must also agree with Dr. Leonard that general debility and deficiency of certain vitamins are predisposing factors. What is yet to be explained is the fact that the disease may attack a healthy mouth in a healthy individual, for this is often the case. Many times we find an acute Vincent's gingivitis in what one might call a normal mouth with none of the aforesaid predisposing factors, either local or systemic, present.

It is not so difficult to account for the frequency with which patients develop recurrent attacks of the disease. Unsanitary mouths, which include those rendered unsanitary by neglect, and those where patients are unable to properly care for periodontal pockets and the like, unquestionably predispose to a recurrence. Local areas of irritation, particularly around partially erupted third molars, are also contributing causes. In other words, if an acute Vincent's infection has once been successfully treated, the likelihood of a recurrence is infinitely less in a mouth free of irritation, free of pockets, and free of deposits and food debris.

Dr. Leonard speaks of the "severe acute (type) which unless properly treated may lead to death in a few days or weeks." Personally I have never encountered a death from Vincent's infection. Many years ago such a case was reported in this city, but the post-mortem findings were not conclusive.

As Dr. Leonard says, nearly everyone has his own special remedy in treating this disease. I feel that the sooner the irritating deposits and collections of food debris can be removed, the quicker will be the cure, and where patients present such conditions, I do not hesitate to remove this foreign matter at the time of the first visit. This is followed immediately by an application of 10 per cent chromic acid to all ulcerated areas, and after thoroughly rinsing the mouth with water, an application of 25 per cent silvol solution is made to the entire oral cavity, after which the patient is not allowed to rinse the mouth. The patient is given a sodium perborate wash and a potassium permanganate wash to be used alternately every hour. Quantities of fruit juices are advised, and if the patient has a substantial rise in temperature, he is ordered to bed. In most cases the relief from pain occurs rather promptly, and on the second day another application of chromic acid and silvol solution is made. At subsequent visits further measures to improve mouth sanitation are employed, but no further application of chromic acid is advisable unless new ulcerated areas appear. The mouth washes are continued until the infection is well under control, though the intervals for using them are lengthened. This treatment is the most successful in my hands, but when cases do not respond, other agents are employed. Injections of neoarsphenamine are advised only when all other methods fail.

There has been much criticism of the use of 10 per cent chromic acid because, it is said, this agent destroys gingival tissue. It is very doubtful that more tissue is destroyed by this agent than would be destroyed by the infection if it is not promptly controlled.

While patients with "general debility" certainly need proper therapy, I do not believe, as Dr. Leonard says, that such debility "is the essential cause of the disease."

HARRY J. LYONS,
Richmond, Virginia

Dr. Leonard's "Initial Statement" presents an interesting appraisal of an important dental and medical problem. Many aspects of the disease are still the subjects of controversy, indicating the incomplete scope of

our knowledge concerning the problem. Dr. Leonard makes a number of statements with which I would take issue. Debate on all these issues would take up more time and space than appears permissible in this project. These debatable statements indicate the need for further study and observation of the disease.

While our knowledge of the disease is still incomplete, enough factual information concerning its causes and clinical course is known for a fully rationalized approach to its treatment. Despite this, there exists a chaos regarding the therapy of this disease which in itself presents a difficult problem. Differences of opinion on this topic have given rise to marked antagonisms between the several schools of thought. One might justifiably wonder why, after twenty-five years of intensive consideration, a fairly well standardized treatment has not been evolved and generally accepted. There are probably many answers to this question. One stands out as an indictment against the profession. It is the failure to critically and scientifically evaluate the numerous therapeutic methods advocated, particularly the various drugs suggested. This is not a simple or easy thing to do. Instead, too many have taken the easier road of the *post hoc* type of reasoning in considering therapeutic methods. This is by no means a valid method of evaluating the therapeutic usefulness of an agent. It savors of *testimony* and leads one to substitute testimony for *scientific evidence*. In the use of *post hoc* reasoning here, one tends to overlook the fact that all acute infectious nonfatal diseases resolve themselves completely or else subside to chronicity in a short time in the absence of any treatment. The application of a remedy in a case of acute Vincent's infection followed by a subsidence of its severity or its symptoms, does not indicate that the remedy has any therapeutic merit unless the results are compared with carefully established controls. Furthermore, when two or more remedial agents are simultaneously used, as is frequently the practice in treating this disease, neither can be credited as meritorious unless they have been first applied singly and compared with valid controls. These principles are widely ignored in the literature reporting on this problem. There has been too much testimony based on questionable observations made without use or advantage of valid controls. The need is for more scientific evidence concerning the merit of advocated remedies particularly drugs.

The Council on Dental Therapeutics of the American Dental Association has given much attention to this subject. Demands that promoters and marketers of drugs, claimed as being useful in the treatment of Vincent's infection, present scientific evidence rather than testimony, met with no acceptable responses, despite the liberal period of time granted for this purpose. Accordingly, the Council on Dental Therapeutics has deleted from its text—*Accepted Dental Remedies*—all the arsenical preparations and definite anti-Vincent therapy claims for the dyes, chromic acid, bismuth compounds, oxygen liberating compounds such as sodium perborate, and a number of other drugs.

With reference to the method of treatment favored by Dr. Leonard, I take issue with his testimony in support of oxidizing agents and mercuric bichloride. Again, where is the scientific evidence in support of their use? What evidence is there that the acute symptoms would not subside as quickly if the oxidizing agents and mercuric bichloride were not used? Dr. Leonard's statement to the effect that swallowing traces of a mercuric bichloride mixture appears to be harmless is one of questionable merit.

Dr. Leonard admits that ". . . cleaning out the lesions by instrumentation and starting toothbrush procedure is promptly effective . . ." but states that this treatment ". . . is unduly painful and may possibly be dangerous due to bacteremia." The bugaboo of bacteremia needs to be debunked. Despite the wide use of this so-called manipulative method, no cases of grave complications or deaths from its use have been reported. It has been established that a transitory bacteremia follows the extraction of teeth and, more recently, that it may even follow the use of the toothbrush. The statement that the so-called manipulative treatment is ". . . unduly painful . . ." is open to debate, particularly since the appraisal of pain is so difficult and in view of the admission that the method is ". . . promptly effective . . ."

It appears advisable to discuss at some length the so-called manipulative method for the local treatment of Vincent's infection. Too often, it is dismissed by defining it as "cleaning the teeth." It is much more than that. It involves opening, cleansing and aerating the focal incubation zones. It further involves irrigation of these zones from their "bottoms upward" by the hemorrhage that necessarily accompanies it. A more vigorous circulatory response may be induced by the instrumentation. Most important, nature's ideal dressing in the form of a blood clot is left in the region. The manipulative method is accurately defined as "débridement" about which so much is now being written in connection with the treatment of war injuries and infections. Dentists need to acquaint themselves with the principles involved and its technic.

It is important to emphasize that the manipulative method can easily be tested for merit by comparing the responses of comparable lesions in the same case to treatment by this method in comparison with others. Such comparisons are most enlightening. (Author's data not yet published.) In those cases not characterized by such etiologic agents as fatal blood dyscrasias, therapeutic responses to the manipulative method, skillfully applied, are dramatic. (Kodachromes of a typical case supplied to Dr. Leonard for inspection.) The method so changes the environment of the incubation zones and so disrupts the mechanism of the disease as to promptly abort it. In this connection, an important and illuminating analogy may be cited in the case of an acute apical abscess. Irrespective of what drug one may apply topically to the surrounding tissues, such an acute infective process will run its full course significantly unaffected and subside to chronicity when it ultimately establishes a mode of drainage. In running its full course, much tissue may be destroyed. However, this lesion can be promptly aborted by a manipulative method which involves the establishment of adequate drainage through the tooth crown. Establishing drainage through the crown probably does other things as well, such as altering the oxygen tension in the pulp canal. Here is a manipulative method that is promptly effective and the merit of which may be confirmed by the ability to reverse the process at will, by simply closing the crown orifice. The environment of the incubation zone is effectively altered by this method, something which cannot be done by the topical application of drugs to the exterior of the oral tissues surrounding the diseased tooth. The suggested manipulative treatment for acute Vincent's infection acts in a comparable manner. It's basic merit and all-important indication for use is that it promptly stops the tissue destruction characteristic of the disease. By comparison, under the so-called drug therapies the acute stages of the disease subside in several days by virtue of the disease itself changing the environment of its incubation zones, sloughing itself out into the open and destroying its incubation zones. The resultant deformities of the interdental tissues are characteristic and too well-known to dentists to warrant description. The development of these deformities under treatment in cases seen early in their course is one of dentistry's unpardonable sins in light of our newer knowledge.

Dr. Leonard's suggestions on the indicated general supportive care are commendable. He failed to discuss the relation of tobacco smoking to the problem which many believe to be important. Many other aspects of the problem might be debated if space permitted.

SAMUEL CHARLES MILLER, D.D.S.
New York, N. Y.

I would prefer the term "Necrotic Gingivitis" as suggested by Box so that the etiology is not related primarily to the Vincent's organisms. It is my opinion that the infectious aspect should be de-emphasized. This is borne out by a sentence in the "Initial Statement,"—"However, it has not been found possible to introduce the infection from the most severe case into the gingiva of a perfectly healthy individual." Those cases which end fatally most likely have another disease in addition to the Vincent's infection, the former being the cause of the lowered resistance creating the soil for anaerobic bacterial growth. Leonard's statement, "Some patients with mild chronic Vincent's infection are able to make the symptoms disappear or reappear at will by taking niacin or not taking it," strengthens this opinion.

We have found 50 per cent hydrogen peroxide too strong, producing lingua filaceosa chromatica (furry tongue), just as sodium perborate does. We use only the 3 per cent commercial hydrogen peroxide diluted, 1 teaspoonful to 4 ounces of water.

The constant reference to "this infection" I believe is an error. The employment of chromic acid, is based on a false premise. This acts simply as a caustic harmful to tissue and tooth structure alike. The injection of arsenicals, in spite of *apparent* successes reported, should never be used for typical necrotic gingivitis because of the danger of liver damage. Constant irrigation (even with plain warm water), attention to general health and nutritional fortification, cleansing of the mouth and teeth (removal of all irritants), and restoration of correct function are the surest means of eliminating this disease without undue tissue destruction.

I agree heartily with Leonard that, "It should not be enough merely to relieve the patient of Vincent's infection. The underlying cause should be discovered and eliminated if possible, for if not, the patient will sooner or later have other and perhaps more serious impairment of health."

RUSSELL A. SAND, LT. COL., U. S. ARMY D. C.
Clinton, Iowa

In his "Initial Statement" on Vincent's infection, Dr. Leonard has briefly but adequately covered the basic principles of etiology, bacteriology, diagnosis, and treatment of a condition which has proved to be a confusing, incompletely understood disease. In spite of the vast experimental and clinical research in etiology and treatment, present day investigators admit they do not yet know the complete answers.

Vincent's infection may truly be called a disease for which there are many cures. Some curative measures may not be as efficacious, as rapid, or as complete as others, and yet we know that thousands of cases have been successfully treated by methods and drugs as different as a local anesthetic is from a general anesthetic.

The etiology has been responsible for almost as many theories as has the treatment, but scientific research has not yet proved to the satisfaction of the careful investigator that the bacteriology of the disease is fully understood. Because of the ease of clinically diagnosing the acute phase of the disease, we might say that the diagnosis is at least a matter of proof, even though etiology and treatment are not. However, we have only to point to the false conclusions which may be drawn from positive or negative smears for microörganisms known to be associated with the disease to impress upon us that even diagnosis may be a questionable problem except in the mildly acute state.

Regardless of the incomplete, indefinite knowledge of all phases of Vincent's infection, it can be said that certain basic fundamentals in the study and management of the disease are fairly well proved.

Many factors are known to play an important rôle in the etiology of Vincent's infection:

1. Lowered resistance of the gingival tissues due to local factors such as calculus, caries, ill-fitting crowns, bridges, and fillings, erupting or malposed teeth, investing tissue pathology such as periodontoclasia, etc.
2. Lowered tissue resistance caused by systemic and organic disease or by glandular disturbances.
3. Lowered tissue resistance caused by nutritional deficiencies most important of which are the various avitaminoses.

Dr. Leonard's point that there are all degrees of severity of this infection is of great importance because many cases of subacute to chronic Vincent's infection are confused with other types of gingivitis or with periodontoclasia. By the same token, many cases of gingivitis have erroneously been diagnosed as Vincent's infection in the absence of cardinal symptoms of the acute disease.

There has been a great deal of discussion pro and con on the communicability of the disease. There is, I believe, little doubt that the disease is communicable under certain favorable conditions. Many have made the statement that an individual with a clean, healthy mouth could never develop Vincent's infection. This is true to a very great extent, but we must assume that the clean, healthy mouth referred to means one that shows no lowered resistance, no local irritation, and no nutritional deficiency, but does show a high degree of care. The chances of such a mouth developing the

disease even on exposure, are remote, but by no means does this disprove the communicability of the disease any more than not developing tuberculosis on contact disproves the communicability of that disease.

The low incidence of Vincent's infection in the armed forces in the present conflict supports the theory that proper mouth care and diet play an extremely important part in the etiology of the disease.

Despite the lack of standardization of treatment and specific therapeutic agents, Vincent's infection has been very successfully treated by the means Dr. Leonard has mentioned in his "Initial Statement." The method of choice should be the one most successfully used by the individual operator. Care should be exercised to use medicaments which cause a minimum of tissue destruction while producing the maximum effectiveness in combatting the disease. Chromic acid or any caustics should be used in the maximum dilutions which will produce the desired results.

In my personal experience, arsenicals used topically in the powdered form over all affected areas, previously isolated with gauze or cotton rolls, has proved the most effective treatment for very acute cases. It is not as effective in the sub-acute or chronic cases. The arsenical is allowed to remain in contact with the tissue from 5 to 15 minutes, after which the patient is given a mouthful of warm water with instructions to swish it around his mouth for two or three minutes. He should not rinse his mouth, drink, or eat for a half hour thereafter. This office treatment is augmented by sodium perborate mouthwash at home—a level teaspoonful of perborate to one-half glass of warm water—used after each meal and before retiring. During these intervals, between sodium perborate rinsings the patient is instructed to rinse his mouth thoroughly every hour with warm water to mechanically eliminate any organisms and debris which cling to the teeth and soft tissues. Usually two or three office treatments on succeeding days, and three or four days of home treatment will eliminate the acute phase of the disease.

I prefer sodium perborate to hydrogen peroxide as a more effective source of oxygen to combat the anaerobic organisms. However, a word of warning should be injected into any discussion of its use since we have all observed mucous membrane burns resulting from the prolonged, injudicious use of too strong solutions of sodium perborate. It should only be used on prescription and careful instruction by the dentist and the length of administration carefully controlled.

Parenteral administration of arsenicals in the treatment of Vincent's infection has not proved too effective in my experience. The conclusion we might draw from this observation is that the bacteria, while not yet definitely identified, may be found to vary in different types of cases.

Like Dr. Leonard, I have seen uncontrolled Vincent's infection in mouths of individuals who have been undergoing maximum anti-luetic treatment for long periods of time. The most effective parenteral administration of arsenicals I have seen has been in very young children where local topical treatment of any kind could not be carried out effectively.

Too much stress cannot be made on treating the acute phase of the disease as an acute disease. There should be absolutely no use of the toothbrush, nor should any attempt be made to remove local irritants such as calculus until the acute manifestations have subsided. The patient's elimination should be carefully checked, large amounts of fluids should be forced and the use of tobacco, condiments, and alcohol should be restricted. A bland diet, low in carbohydrates and high in vitamins, especially B and C, should be instituted. Vincent's infection should never be regarded lightly and we should bear in mind at all times in the treatment of this disease that serious, dangerous results may occur from extension of the disease to the throat and air passages.

BENJAMIN TISHLER, D.M.D.
Boston, Mass.

The following comments are my reaction after reading the "Initial Statement" on Vincent's Infection:

I believe that strictly speaking Vincent's infection is an acute ulcerative condition of the gingivae.

I have not noted an unusual sensitivity of this tissue when treating periodontal pockets in which large numbers of the microörgan-

isms were observed microscopically.

No reference was made in the statement to excessive smoking which I have found to be an important debilitating factor in most cases that have come under my care.

My experience led me to believe that it is most prevalent in young individuals; cases observed in patients over 35 have been rare, therefore, I feel that a resistance is built up as persons grow older.

1 have found that, contrary to the statement, it will develop occasionally in well cared for mouths.

My treatment is very simple, (1) no smoking, no alcohol, no condiments, (2) a pint of orange juice daily, (3) a topical application of 1 per cent acri-violet solution applied to every interdental papilla, (4) a home mouth wash of sodium perborate used every half hour (1 teaspoonful in a half glass water, held in the mouth for three minutes). I do not believe in rinsing; it is not thorough enough. Should a mouth burn develop, solution is diluted one-half strength.

The interval between visits is increased as improvement is noted; scaling is done at this time.

Cases should be healed in two weeks unless some unforeseen complication is present.

CHAS. H. M. WILLIAMS, D.D.S. B.SC. (Dent.)
Toronto, Ontario

The "Initial Statement" is excellent and this reply will, in the main, support the opinions expressed by Dr. Leonard.

It has been our effort both in practice and in the teaching of periodontology at the University of Toronto to stress main principles rather than details of treatment. A very condensed form of the teaching in respect to Vincent's infection or necrotic gingivitis follows.

INTRODUCTION

"The resistance of the part is of greater importance than the infectivity of the organisms." (Tunnicliff). Efficient treatments are therefore based mainly on the plan of raising the resistance of the affected tissues rather than on eliminating bacteria by the topical application of drugs.

ETIOLOGY

The predisposing causes are more important than the exciting causes both from the standpoint of prevention and treatment. Failure of healing in some mouths or in certain areas of some mouths is usually the result of some local or systemic condition that delays healing rather than the presence of particularly virulent organisms. The predisposing causes are both local and systemic. The local causes are usually more important than the systemic causes with individuals of average general health. The latter statement is based on clinical experience for this area and is supported in part by the results of a study on groups of humans of good general health who were, by habit, consuming a diet suboptimal in vitamin C and for whom the administration of vitamin C in large amounts over a six months period was negative in effect on the gingivitis each person exhibited. However, the local and systemic predisposing causes of Vincent's infection will be found to vary in relative importance for different groups of people in the same area and for different localities. Reports from Great Britain during the last two years seem to indicate that mild scurvy is now a fairly common predisposing cause of Vincent's infection in that area.

The exciting cause is not definitely known. The Vincent's organisms have sometimes been considered the bacterial agents responsible for the infection but some authorities now believe that these organisms are nonpathogenic saprophytes and that an increase of their number in an area is a result of the disease rather than the cause. The significance of an accompanying anaerobic streptococcus has lately been suggested. The use of the bacterial smear as a diagnostic aid is not stressed and diagnosis is accomplished mainly by use of the history and by clinical examination.

SYMPTOMS

No discussion is offered.

TREATMENT

The treatment should be divided into two phases. The first and introductory part is designed to bring the painful acute ulceration under control. The *second and much more important phase* is designed to complete the elimination of the infection and to control the common tendency for the acute ulcerative stage to recur. No matter

what treatment is employed for the acute ulcerative stage a thorough elimination of the incubation zones as a second phase of treatment is necessary if the result is to be lasting. It is a disease of unhygienic crevices and depressions, and it is not usually found on the self-cleansing surfaces of the soft tissues. If therefore the crevices and depressions can be established and maintained in a relatively hygienic state, the infection automatically ceases to exist. Introduce office and home procedures aimed at the establishment and maintenance of hygienic conditions in the crevices as soon in the treatment as the comfort of the patient will allow.

GENERAL PLAN OF TREATMENT—
PAINFUL ACUTE ULCERATION

Office

Remove debris from surface of ulcers either by employing an effervescent drug such as hydrogen peroxide or a proteolytic agent such as caroid and then either cauterize ulcers with Churchill's iodine and 35% silver nitrate, or cover ulcers with cement pack as lately described by Box. *(Necrotic Gingivitis*—Canadian Research Foundation, Bulletin Number Twenty-Five.) If caustic drug is employed apply with tiny pellets to *ulcers only*, and a minimum number of times (usually once or twice) at forty-eight hour intervals.

Home Care

Hydrogen peroxide 3%, one part to three parts warm water as mouth-wash six to eight times daily for two or three days, (or mouth-wash described in "Initial Statement").

SUB-ACUTE CONDITION

Office

Scaling and other procedure for the removal of irritants, except extractions, should be performed as soon as the comfort of the patient will permit. Polish teeth with pumice and rubber cup. Either instruct patient to clean interproximal spaces frequently with interdental cleansers or cleanse spaces and fill with therapeutic cement pack previously referred to. The above procedure may be used to further reduce swelling, tenderness, and bleeding, before proceeding with operative elimination of secondary incubation zones. The use of interdental cleansers is very beneficial after ulcerative destruction of the inter-proximal gingival papillae has caused the establishment of unhygienic interproximal spaces. The following routine is simple and efficient. Patient is instructed to dip interdental cleanser into hydrogen peroxide 3% before using in each interproximal space and this procedure is to be repeated after each meal and at night until remaining tenderness and bleeding is obviously reduced, usually at the end of three to four days. The use of the hydrogen peroxide should then be discontinued but the use of the interdental cleanser at least twice daily during the weeks or months until the papillae are re-established, is very important.

A thorough clinical and radiographic examination should be performed as soon as the tenderness and bleeding are reduced and a plan of treatment should be arranged for the elimination of local and systemic causes of lowered resistance of the gingival tissues. This most important phase of treatment constitutes the dentist's main responsibility to the patient. Appointments for observation of healing and minor treatment as indicated should be at one to three-month intervals over a period of six months to a year, until the main aim of treatment is attained—maximum reëstablishment of healthy, interdental papillae.

WILLA YERETSKY, D.D.S.
Grand Rapids, Michigan

A discussion of the treatment of any disease presupposes a correct diagnosis of that disease. However, there is probably no other disease in the list of afflictions of the human species in which the diagnosis is so frequently mistaken and the cure of the disease thereby prolonged.

The classic, clearcut description of the lesions as described in the "Initial Statement" is never difficult to recognize. The fact that the disease occurs in tissue which has become debilitated from other causes makes it possible for it to become a complicating factor in other infections of the oral cavity. Not infrequently, these other infections are unrecognized and the entire syndrome is labelled "a typical Vincent's infection."

The most common acute infections of the buccal mucosa are those with *Streptococci,* usually the *viridans* but also other types; and with the *Monilia.*

In general, if a patient presents with a temperature of 100 F. or more, and an acute infection over the entire mucosa rather than with lesions limited to the gingival margin, a mixed infection should be suspected.

The patient is frequently extremely ill. The lesions of a *Streptococcus* infection are not so deeply penetrating as in the Vincent's. The ulcers are discrete, small vesicles which rupture early leaving a grayish membrane, which, unlike the necrotic membrane of a typical Vincent's infection, cannot be so easily wiped off. These ulcers may be very generally distributed over the pharynx, buccal mucosa, and gingivae. The patient will have a severe lymphadenopathy of the cervical region, also.

It is possible that the *Streptococcus* is the invading organism in these cases and, because of the injury and change wrought in the tissues, the Vincent's organisms become secondary invaders; but of equal importance. This form of the disease is very contagious, especially in children.

In this type of infection, sulfonamide therapy is of great importance. Sulfamerazene has the advantage of requiring only one half the dosage of the other sulfonamides. It is considered less toxic for this reason.

Therefore, the patient is placed upon sulfamerazene therapy by mouth for three days. This reduces the fever and controls the cocci infection.

However, the sulfonamides do not (nor incidentally, does penicillin) have any effect upon the Vincent's organisms.

It has been found that if Vincent's therapy is instituted locally at the same time that the sulfamerazene is begun, the course of the disease can be so shortened as to be extremely dramatic in the change. Three to five days of treatment will be all that is necessary for a cure.

The mouth is first irrigated with tincture of metaphen. This has a deterrent effect upon the cocci and also, upon any *Monilia,* if present. After irrigating with tincture of metaphen the mouth is dried with sponges or cotton rolls, which are left in place during the application of the next drug. This is an emulsion of neoarsphenamin in glycerin, which in my hands, has proved most successful. A .45 gm ampule of neoarsphenamin is opened just prior to using it and emulsified with one cc of glycerine. It must be emphasized that this is always made fresh just before using. It is never allowed to stand. This is applied by means of a wire loop directly upon the lesion. The loop has the advantage of being useful to remove the overlying necrotic material (in which there are no *Spirocheates)* and then planting the medicament directly on the ulcerated surface. Relief is very rapid under this treatment.

There is no rational basis for using any of the arsenicals intravenously, since relief and cure is obtained much more rapidly by local means. It is true that an intravenous injection of neoarsphenamin will relieve or even abate the symptoms. But since so many other factors, such as subgingival calculus, can prolong it, it is more logical to apply the drug locally, at the same time removing the calculus, or other irritating factors.

For home care the patient is given a potassium permanganate solution, in the strength of 1:1000 to use as a mouthwash every three hours. He is also told to use hydrogen peroxide as a mouthwash one hour after the potassium permanganate. This is in the full N.F.S. strength. Frequent irrigation reduces infection and stimulates the circulation.

This commentator has had no experience with bichloride of mercury used in conjunction with peroxide and would hesitate to do so because of inherent dangers in accidents with it. There are so many other drugs which are as effective and to which no danger is attached.

Aside from this all statements in the "Initial Statement" are substantially acceptable.

Concluding Statement

It is interesting to note that the only medically trained dentist in the group, Dr. Loeb, who might be expected the most of any to emphasize systemic debility as a cause of Vincent's infection, is the only one who states that he does "not believe that such debility is the essential cause of the disease." It may be that his criticism

is of the word "debility" which means "a loss of strength." I used the word to mean a physiologic loss of tissue resistance which is perhaps not a common medical use of the term. I am convinced that the essential cause of Vincent's infection lies in the chemistry of the tissues rather than in the microörganisms themselves. The preponderance of Vincent's gingivitis among young men who are under mental and physical strain, and its close correlation with niacin deficiency as determined by animal studies as well as by diet studies by myself and others, are quite convincing to me. However, the last word has certainly not been said along that line.

In connection with the word "infection" it should be noted that there is practically no evidence available that the microörganisms of Vincent's infection penetrate the living tissue. These microörganisms apparently live on tissue exudate and necrotic material from which they generate such locally poisonous products that the adjacent living cells are killed. Local treatment then, should rather be "débridement" than antisepsis. The fact that the microörganisms are outside and not within the tissues may explain why they are so little affected by sulfonamides, penicillin, and injected arsenicals.

The lack of uniformity of opinion on etiology, bacteriology, chronicity, and philosophy of treatment, which is brought out by this discussion, emphasizes anew the need for fundamental research on the physiology and bio-chemistry of tissue. The gingival tissue because of its accessibility and its susceptibility to certain deleterious influences should prove a particularly valuable avenue for such a study.

—*Harold J. Leonard*

A Letter to the Editor

"Nutritional Factors Relating to Gingival and Periodontal Health and Disease"

I HAVE read with great interest the discussion of the problem, stated under the above title, in the Supplement to the January, 1944, issue of the Journal of Periodontology. May I be permitted to say a few words, not only relating to the above article, but also to the problem of periodontal terminology.

It is most confusing that the participants of the discussion use the same term to designate different pathologic conditions and different terms for the same pathologic entity. The term "periodontoclasia" is used in one sentence by *Leonard* in his initial statement to designate an inflammatory disease, and as something else in another, differentiating it from "gingivitis." H. *Becks* speaks of "periodontoclasia" as being synonymous with "paradentosis," which he considers not to be of local but of systemic origin. D. *Bell* speaks of "systemic types of periodontal cases" and of "local inflammatory types of periodontoclasia." *Bricker* speaks of dental periclasia." *Francis* seemingly uses the term "periodontoclasia" as a general term. Dorothy *Hard* uses the term "gingivitis" and "periodontoclasia" or (periodontal disease) without stating what is meant. The same is true for Dorothea F. *Radusch* who uses the term "periodontoclasia" as a general term for all periodontal disturbances. Sinclair speaks of "diffuse atrophy" as applied to the types of periodontal cases which Becks calls "paradentosis." Willa *Yeretsky*, most probably aware of the confused nomenclature, speaks only of "gingival health." just as *Ziskin* speaks only of gingival and periodontal health and disease."

The use of these different terms, with such varying meaning, is certainly most confusing and I think it should be avoided. If the recommendation of the Nomenclature Committee of the A.D.A., as well as the Academy of Periodontology, have any meaning they should be followed in the official organ of the Academy. Anyone submitting a paper to be published in the Journal of Periodontology should be compelled to use the terms accepted by the Academy and the A.D.A. If the author does not use the proper terms the editor should be authorized to change these terms—after consultation with the author—in accordance with the recommendations which have been established. If this policy is not strictly carried out we will never come to a proper understanding. It is the duty of the nomenclature committees to check and make recommendations for future changes in nomenclature in accordance with developments in this special field.

May I say a few words with respect to Nutritional Factors in Periodontal Pathology: The only one with whom I could agree in this discussion is Ziskin and I do it from the viewpoint of a practicing periodontist who, as may be known, is not averse to the scientific approach. I have practiced periodontal therapy since 1924 and ever since have been on the lookout for the systemic factors (including nutritional) in this disease complex. I am also daily confronted with the treatment of periodontal cases. I must confess that in all these twenty years I could never put my finger on one single case which was cured by systemic treatment alone,

without proper local treatment, but I have seen innumerable cases which have been cured (arrested) by local treatment without any systemic medication or nutritional correction. This, of course, does not make me believe that the etiology of periodontal diseases is of a local nature alone; it only shows that without proper local treatment we cannot expect good results. From the discussion one gets the impression that a diet analysis or medical examination is the first thing a periodontist should have done for his patient, even before he looks into the mouth and makes a proper diagnosis. This would be just as foolish as to ask the same thing from a patient with a fractured arm before a splint is made. The fact that we have to repeat treatments every three to six months, by no means indicates that the treatment was unsuccessful or that the disease reoccurred; we do it to prevent reoccurrence. As long as systemic treatment or dietary methods cannot prevent the formation of soft and hard deposits on the teeth and in the gingival sulci it is necessary to remove these deposits to prevent irritation. The dental profession cannot be condemned—not even the periodontists—because they apply local treatment as long as nutritional and systemic research men do not offer a more positive basis for their claims. I do not believe that the medical profession is condemning itself because they cannot prevent the occurrence of appendicitis and they do not investigate the causes before they remove the inflamed appendix.

That *one* single subgingival curettage does not cure a case, does not mean that the local irritation is of secondary importance—in spite of this it might be of primary importance. It is only natural to believe that there is not only an external cause for a disease but there are always a combination of external and internal factors in varying proportions. A fracture of an arm is most often due to an external factor. However, we know that internal factors may play an important role. The bone might be porous due to one of several factors which would make the bone more fragile. Also the process of healing of a fracture is greatly influenced by systemic and nutritional factors. The same can be said of periodontal disturbances. We can consider an irritation by calcareous deposits as a local factor. However, the cause of the calculus formation is an internal systemic affair. In some individuals deposits never form and in others they form very rapidly. When research finds the basic factor for this it will open up tremendous possibilities. Furthermore, we must not forget that the reaction of each individual to calculus irritation is different, and that a systemic disease may modify and increase this variability. We do not need experimental proof to substantiate this statement; investigations as to qualitative differences are, however, much in need.

In the discussion on the nutritional factors, we are told that the diet of people will periodontal disturbances is inadequate in proteins—calcium, iron, phosphorous, vitamins, etc. We are even told how many, out of every ten individuals, are deficient in everything. However, it was not stated whether this is true for periodontal cases only, or for other abnormal conditions as well.

As a practicing periodontist, I maintain that establishment of local hygienic conditions, elimination of gingival pockets, treatment of chronic granulation tissue, establishment of balanced occlusion, elimination of localized occlusal trauma, teaching the patient proper home care to maintain the established oral hygiene, are the main functions of the periodontist. After all this has been accomplished we should refer the patient to the proper authority to investigate systemic conditions, nutritional deficiencies, etc. If the patient presents obvious symptoms of systemic involvement, or if we suspect some systemic disturbance, we should of course refer the patient to the physician. There are cases of ulcerative gingivitis which will not promptly react to local treatment, also a hypertrophic gingivitis of unusual characteristics, etc. Cases of "periodontosis," in which a systemic background is a basic factor, should always be investigated. But even then, the local symptoms should be cared for at the same time. Again and again I have seen cases where systemic investigation did not reveal any disturbance, and nothing was done to correct any nutritional deficiencies and where the local condition was cured and remained cured for many years. On the other hand I have seen cases which were thoroughly investigated and treated for systemic causes or nutritional deficiencies, but the calculus deposits were not removed, and the mouth remained in poor hygienic condition.

In summarizing I would strongly emphasize that local treatment is of first importance, not because the etiologic factor is a local one, but to establish a local cure. The investigation of systemic factors is up to the physician. His responsibility is also the investigation of a nutritional deficiency and the establishment of a proper nutritional regime. As periodontists we can expect great benefits from such coöperation but we should never expect to bring about a cure by systemic treatment alone. Neither should we too quickly blame systemic trouble or nutritional deficiency if we fail locally, as many failures are due to improper local treatment.

—BALINT ORBAN

(h) Vincent's Infection (ABSTRACT)

(Continued from 13(2):118. July, 1942)

240—Stammers, A. Frank: **Vincent's infection: observations and conclusions regarding the etiology and treatment of 1017 civilian cases.** *Brit. D. J.* **76(6)**:147-155. Mar. 1944.

In June, 1941, all Vincent's infection cases attending the Birmingham, (England) Dental Hospital were placed under the author's sole supervision. Complete case histories, medical and dental, were recorded. A total of 1017 cases were admitted in a period of eighteen months—503 males, 514 females; 947 between ages of 14 and 40.

Among the general conclusions drawn from this experience were: 1. While there is a tendency to seasonal variation it is not of necessity constant but can be influenced by such factors as excessive fatigue or anxiety, as in war zone communities. 2. Importance of contagion has been greatly exaggerated, yet its possibility must not be overlooked. Healthy individuals with *really healthy mouths* are comparatively immune to Vincent's infection. 3. "That numerous virulent (shown by intense staining) Vincent's organisms can always be demonstrated by suitable stains in acute cases, but in subacute cases the organisms are scanty and are difficult to find owing to their poor staining qualities." 4. That the fuso-spirochetal infection although primarily responsible for the ulceration is of *secondary* importance in the *inception* of the disease; local predisposing conditions of *paramount* importance; systemic factors merely *contributory* to the aggravation of the local trauma. 5. That hypovitaminosis does not account for increased incidence of Vincent's infection. 6. That excessive smoking plays a part in lowering the local resistance and retarding recovery. 7. Both sexes now equally affected.

The local factors are listed and discussed; constitutional factors mentioned were frequent colds, chronic catarrh, recent illness, operations, and pregnancy, overwork, lack of exercise, and possible vitamin deficiency.

—*G. R. S.*

Announcement

THE FIRST open meeting of the New York Institute of Clinical Oral Pathology will take place in Hosack Hall at the New York Academy of Medicine, 2 East 103rd Street, New York City, on Monday evening, October 30, 1944, at 8:15 P. M.

Dr. Arthur H. Merritt will preside at a symposium on "Fluorine in Dental Public Health."

The following outstanding investigators will participate:

DR. FREDERICK S. MCKAY
Colorado Springs
"Fluorine and Mottled Enamel: A Historical Survey"

DR. H. TRENDLEY DEAN
Senior Dental Surgeon, U.S.P.H.S., Bethesda, Maryland
"The Epidemiology of Fluorosis and Dental Caries"

DR. WALLACE D. ARMSTRONG
University of Minnesota (Associate Professor of Physiological Chemistry, Director of Laboratory of Dental Research)
"The Fluorine Content of Enamel in Relation to Resistance of Teeth to Caries"

DR. BASIL G. BIBBY
Dean, Tufts Dental College
"Effects of Topical Application of Fluorides in Dental Caries"

DR. DAVID B. AST
Assistant Director for Oral Hygiene State of New York Department of Health, Division of Maternity, Infancy and Child Hygiene
"The Practicability, Efficacy and Safety of Fluorinating a Communal Water Supply Deficient in Fluorine to Control Dental Caries"

For further information address all communications to Executive Secretary, 101 East 79th Street, New York 21, N.Y.